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THE AQUEOUS HUMOR IN GLAUCOMA*

A. MAGITOT, M.D.
PARIS, FRANCE

It may appear somewhat bold to renounce old creeds, but no progress can be made in any specialty unless one seeks to interpret clinical facts in the light of the fundamental principles which present day physiology puts at one's disposal.

For fifty years we ophthalmologists have been building theories on erroneous ideas as far as the ocular fluids are concerned. We have frequently forgotten that the eye, although a highly specialized organ, is undoubtedly subject to the same general laws which govern the whole human organism. When we have thought about the ocular circulation we have neglected to apply to our study the information which the physiology of the general circulation has taught us. We have falsely, it seems to me, preferred to consider the circulation in the eye as in no way resembling the fluid exchanges which occur in other regions of the body.

For twenty years I have devoted much time to the study of the aqueous humor. From my observations and experience I have arrived at a conception of the pathology of glaucoma which perhaps many will find revolutionary, but these conclusions are the direct consequence of my views concerning the nature and origin of the ocular fluid.

I earnestly hope that since the time when Elliott wrote that I was an "iconoclast whose object it was to upset all our preconceived ideas and to induce us to reconsider the very foundations of our belief," opinions have changed. I shall be greatly pleased to find among my colleagues attentive minds and well informed critics who will give my views their sincere and unbiased consideration.

In 1916 I wrote that the aqueous humor was a fluid issuing from the blood by dialysis. The biochemical researches of the last ten years, notably those of Baurmann, Meesmann, de Haan and van Creveld, Duke-Elder, Gaedertz and Wittgenstein, and Cohen and William, have confirmed this opinion and supported it with proofs which I could not lean on when I first expressed this theory. My first opinion was based on the researches of Mestrezat on the spinal fluid.

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It would be impossible for me in this limited paper to cite all the arguments which emphasize the sanguineous origin of the aqueous humor. These references, which support my theory of the origin of the aqueous by dialyzation rather than by secretion, have been summarized by Duke-Elder and deserve the most serious consideration.

As my first premise I wish to ask the acceptance, at least tentatively, of my belief that the aqueous humor is simply blood serum modified by certain thermodynamic forces. In addition, I wish to insist on another point which I treated of in 1928, in a paper entitled "The Multiple Sources of the Aqueous Humor." As the title suggests, this paper presented the subject in a more general aspect, but in this paper I wish to recall that the aqueous is not necessarily formed from a single source.

In my opinion it is an error to consider the aqueous as a fluid special to the eye, for there exist in the body other fluids with a chemical composition very similar to that of the aqueous, and these various fluids may very properly be considered as belonging to a single group. The cerebrospinal fluid fills the ventricles and bathes the central nervous system just as the aqueous fills the anterior chamber and the meshes of the vitreous and bathes the retina. In this group may also be placed the perilymph and the endolymph of the ear. Leaving the cephalic extremity, one finds in the connective tissue of the amnion of the new-born child a liquid of the same nature.

The cerebrospinal fluid and the aqueous are not only similar chemically, but, as the recent researches of F. Adler and Marx emphasize, these two fluids are still further analogous in their pressure reactions. Under normal conditions there are no other aqueous fluids of this particular nature in the body, but under pathologic circumstances aqueous humors of this same chemical and physical nature may appear in various parts of the body.

Occasionally the interior of the bile ducts becomes desquamated and occluded, isolating the hypertrophied gallbladder from the hepatic secretion. Under the lancet of the surgeon such a gallbladder discharges a content as clear as crystal. This fluid, which has been designated by the name of white gall, is also chemically similar to the aqueous and to the spinal fluid (Gosset, Loewy and Mestrezat). One cannot conclude that this fluid has been secreted by a mucous membrane, for the mucous membranes have disappeared, and the atrophied wall of the gallbladder is reduced to a mere fibrous coat in which can be seen, however, a well developed capillary network. Where, then, but from the capillary network can this fluid have originated?

On the other hand, Achard (1928) observed the same fluid in cysts of the kidney. He was able, in experimental hydronephrosis, to observe the gradual transformation of a secreting cavity into a space

filled by a fluid having all the characteristics of a simple dialysate of the blood.

The contents of hydatid cysts furnish a further example. The taeniae develop in the midst of tissues which seek to wall off the intruders by an enclosing membrane. The hydatid cyst fluid formed by the capillaries of this sack likewise has a chemical constitution almost identical with that of the spinal fluid, while the diffusible substances that it contains are sufficient for the nourishment of the parasite.

Lastly let me draw attention to a fact which has considerable bearing on my theory of the origin of the aqueous humor. The fluid of certain edemas contains only 0.35 Gm. of proteins and 7 Gm. of chlorides per thousand cubic centimeters (Boy-Teissier and Rouslacroix). These figures are very close to those for the ocular and spinal fluids.

It would seem, therefore, that, far from being something peculiar to the eye, the aqueous is in reality a fluid widespread in the body. A humor which exists under normal physiologic conditions and which may also be caused to appear by artificial means must be an important and ever present element in the body economy. Achard has given such humors the name of "lacunaire" fluids, and Mestrezat called them "originels" fluids. They are, it would seem, simply the common tissue fluid identical with the fluid that fills the connective tissue spaces and cellular interstices, and that bathes all the cells of the body and conveys to them the indispensable materials for their nutrition.

It should be remembered that the blood, which contains the nutritive principles and carries off the waste products, is not the true interstitial medium which bathes the cells. Its function in cell metabolism is but an indirect one. The immediate rôle belongs to the fluid which, outside of the closed canalization of the vessels, is in intimate contact with every living cell. Coming from the blood, this tissue fluid takes from its parent the sugar and the mineral elements which are necessary to life, but it is very poor in proteinic substances which are useless as nutritive elements. This interstitial fluid is therefore the true interstitial medium in which the cells live. It assures the fixity of the ionic reactions and of the molecular concentration.

There are striking examples in the body of tissues that are nourished solely by a fluid which contains only certain elements of the blood plasma. One sees this in the growth and nutrition of cartilage. The cartilage cells are enclosed in a solid sheath and can derive their nourishment only by imbibition from the liquid which is intimately in contact with each cell.

We as ophthalmologists are very familiar with a similar condition in regard to the nourishment of the crystalline lens. Deprived of its blood vessels from the moment of termination of the intra-uterine period, it must draw its food from the aqueous. Thanks to this

medium it is adequately supplied, and we know that it increases in size during the first twenty or thirty years of life.

The visual cells also, cones and rods, are separated on one side from the choriocapillaries by the pigment epithelium, and on the other from the capillaries of the retina by the thickness of the external plexiform layer, and yet nutritive elements are indispensable to these highly active nerve cells.

The experiments of Adler provide comparative data for the quantity of sugar in the aqueous fluid in different regions in the ocular globe. In the anterior chamber the aqueous contains an amount of sugar very nearly the same as that in the blood, but in the vitreous there is less and less the nearer the fluid approaches the retina. In my own experience, after an ocular contusion I have observed that the sugar disappears completely from the aqueous, which suggests that the cells, in order to reestablish their energy potential, have consumed all the sugar that was stored in the anterior chamber.

The production of the interstitial fluid takes place by dialysis through the walls of the capillaries. The union of their endothelial cells forms the living semipermeable membrane which, according to physicochemic laws, presides over the osmotic exchanges. The forces which act on this membrane are many and opposing. The crystalloids of the plasma traverse it and balance each other on either side according to their electric charge, carrying with them a certain quantity of water. On the other hand, one must take into account the pressure of the blood stream which has a tendency to drive the water through the membrane, but to this force is opposed Starling's colloid-osmotic pressure, a sort of hydrolyte of the proteins of the blood which attracts the water and restrains its passage. Thus in the normal state, according to Bayliss, the hydraulic pressure in the arterial capillaries being superior to the osmotic pressure of the proteins, some water passes through into the tissues. But when this hydraulic pressure diminishes, the proteins become concentrated and the colloid-osmotic pressure becomes stronger in the venous capillaries. Thus an attraction of the fluid in a contrary direction takes place, resulting in its absorption.

In the eye these different forces acting in opposition to each other give rise to an ocular tension such as is observed immediately after death. I have shown, indeed, that immediately after death, the ocular tension does not fall to zero as Donders believed, but remains at about 10 mm. of mercury, falling to zero only at the end of from fifteen to sixteen hours.

Such observations on the pressure in the eye immediately after death, or on a freshly enucleated eye, show what proportion of the ocular tension is due to local blood pressure and what percentage to the aqueous, that is, to the tissue fluid itself.

I should like once more to reiterate my opinion that the interstitial fluid of the eye does not possess a specialized drainage system; like all the other tissue fluids of the same group it is dialyzed from the arterial capillaries and reabsorbed by the venous capillaries. The movements of this fluid are chiefly regulated by the presence or absence of sugar, which is an urgent and indispensable nutritive substance to the retinal cells; the mineral elements function simply to maintain the physical quality of the medium necessary to preserve cell activity. One can therefore expect, and in reality actually find, the most active and influential metabolic changes taking place in the retina itself which is situated at a considerable distance from the canal of Schlemm. Therefore, from both an anatomic and a physiologic basis it would seem irrational to consider the canal of Schlemm as being only a drain for the aqueous humor.

It is to be noted that the aqueous and the spinal fluid have one more mark of similarity: They are both excellent mediums for the preservation of nervous energy. The laboratory worker must utilize either spinal fluid or aqueous if he wishes, even for a short time, to preserve living nerve cells "in vitro."

In the eye the circulation of fluid is normally very slow. Certain substances remain for a long time in the aqueous humor. It has been pointed out by Czapody that atropine instilled into the conjunctival sac remains in the anterior chamber for several days. It is likewise known that, after puncture of the cornea, an abnormal quantity of chlorides and an increased number of immune bodies are to be found in the aqueous for as long as three weeks (Asher, Morax, Loiseau).

It is sometimes thought that the fluid movement is fairly rapid; this impression arises from certain observations on the injection of dye substances into the eye. Seidel has shown that these dye substances appear almost immediately in the veins, and therefore some conclude that the fluid exchange is rapid, but such a conclusion is a faulty interpretation of what actually takes place under normal conditions. In injecting the dye the eye has been penetrated, and not only is the hydrostatic equilibrium disturbed, but a nervous perturbation has been created. Under normal conditions the exchange of fluids is slow. Liquid is carried away only by the changes in concentration of the fluid which are brought about by the consumption of sugar and mineral elements.

Water can be artificially removed by modifying the colloid-osmotic pressure of the blood. Hertel and Duke-Elder have shown that venous injections of hypertonic solutions attract the water and that it can at will be returned by another injection of a hypotonic solution. Yet normal conditions do not allow of such exchanges. In the living the blood concentration has a remarkable fixity, and researches made on

glaucomatous patients have shown that neither the concentration of crystalloids nor that of colloids has a clinical significance.

AQUEOUS HUMOR AND TENSION IN GLAUCOMA

From what has been stated, it appears, therefore, that the aqueous is but the tissue fluid of the eye. It fills the large spaces, such as the anterior chamber, fills also the meshes of the vitreous and percolates into all the tissue spaces of the uvea and retina, sclera, cornea and optic nerve. Lastly, the pressure under which this tissue fluid is stored up is one factor in the formation of the ocular tension.

Take for example the tension of the eye of a cat or a dog. The reading of the Schiötz tonometer indicates 20 mm. of mercury. If the animal be suddenly killed by means of an intravenous injection and the ocular tension taken again, the tonometry figure will be 10 mm. of mercury. From this simple observation one knows that two factors combine to build the ocular tension, namely, the blood pressure, which disappears as soon as the heart ceases to beat, and the tissue fluid pressure, which has been built up during life and which persists for some hours after death.

The same experiment may be performed on a hard eye which has been enucleated for painful glaucoma. Before the operation the tension is 60 mm. of mercury; after the operation it is 20 mm. This experiment gives one information on the pathologic tension. It shows that the difference between 60 mm. and 20 mm. of mercury is due to the blood pressure and that this pressure is very high as compared with the pressure in a healthy eye. The figure, 20 mm. of mercury, the remaining tension, shows moreover that the pressure of the tissue fluid in this eye is double the normal aqueous pressure.

1. Tissue Pressure. What one observes is this: If the cornea of an eye in which the chamber is very shallow be punctured, one will obtain only a few cubic millimeters of fluid. In cases in which the anterior chamber is deeper, one will be able to extract from 120 to 150 c. mm. of fluid. As the usual contents of the anterior chamber of an emetropic eyeball are from 150 to 200 c. mm., one is led to remark that there is a smaller quantity of aqueous humor in a glaucomatous eyeball than in the normal eye. Yet the high figure for the residual tension in the enucleated globe might lead one to believe that there would be an excess of fluid. This is not so because the aqueous, being a tissue fluid, must be looked for not only in the anterior chamber but in the intercellular spaces. Indeed, it remains imprisoned in the different coats of the eye, around the cells of the retina and uvea and inside the meshes of the swollen vitreous.

In general pathology such an increase in tissue fluid is called edema. Therefore the increased residual hypertension in the glaucomatous eyeball must be considered as a kind of edema.

This opinion is confirmed by clinical observations. It is known that the epithelium of the cornea is raised by the fluid (Fuchs); that the turgidity of the vitreous causes protrusion of the lens and shallowness of the anterior chamber; that infiltration of the choroid pushes forward the ciliary processes and causes paralysis of accommodation. Indeed, retinal edema around the disk after an attack of glaucoma was described long ago.

In my report at the Congress of Amsterdam (1929) and in an article which appeared in the *Annales d'oculistique,* I drew attention to the error propagated by the belief that all the symptoms of glaucoma are derived from the hardness of the eyeball. The mydriasis of glaucoma is not due to the ocular hypertension. Schmidt-Rimpler and Heine were never able to cause dilatation of the iris by experimentally increasing the pressure of the vitreous, any more than Nordenson was able to cause a diminution of the depth of the anterior chamber by these same means. The two symptoms are due to edema. In the case of the pupil, the dilatation of the iris may be attributed to the fact that the nerve fibers are compressed in their passage through the suprachoroidea. There is, first of all, a motor disturbance, then trophic disturbances due to the paralysis of the sensory fibers which cause a degeneration of the parenchyma of the iris.

A hypertension which many ophthalmologists confuse with glaucoma may, however, exist for a long time without causing any decrease of vision (see the cases of Neuhauser, Orr, Nippe, Elsching, Liégard). I observed a man who for six days after contusion of the orbit continued to have a tension of from 60 to 80 mm. of mercury on the Schiötz instrument, without any sign of visual defect, mydriasis, edema of the cornea or spontaneous pulse of the central artery (1918). On the other hand, there have been many cases of glaucoma in which the tension has been lowered by sclerectomy and which continue to progress toward blindness in spite of all therapeutic efforts.

I think, therefore, that von Graefe was right in including with glaucoma the disease which he described under the name of "amaurosis with excavation," and I agree with Elsching when he speaks of glaucomas without hypertension and of hypertensions without glaucoma.

On the other hand, it is curious to note that in glaucoma simplex the aqueous remains chemically normal. It is only in inflammatory glaucoma that it undergoes a modification. But this chemical change is not specific; the aqueous becomes rich in colloids and poor in chlorides, and here is to be found once more the general characteristic of the "plasmoid" humor (Duke-Elder), a transformation which takes place whenever there is vasodilatation in the anterior segment. Thus in glaucoma and edemas generally the fluids undergo these modifications. In red edemas and red glaucomas the fluids are rich in protein

content, while in white edemas the fluids are rich in chlorides and poor in colloids.

As in angioneurotic edemas of other parts of the body, the edema of glaucoma is subject to frequent variations. This explains the curious ocular tension curves, the oscillations from one day to another and the sudden falls sometimes observed between the morning and the evening readings. When the edema is intense the media are turbid; when it diminishes, the media clear up, but the cloudiness rarely disappears completely. All ophthalmologists are familiar with eyeballs which, even after the tension has been reduced, continue to present that infiltration peculiar to the cornea and so easily discernible nowadays with the slit-lamp.

This edema, although it invades all the tunics of the globe, may be predominant either in the anterior segment or in the posterior segment. When it predominates in the anterior segment there is glaucoma with a deep chamber, while predominance of the edema behind the lens produces the usual clinical picture of glaucoma with a shallow chamber. One might therefore very properly speak of posterior glaucoma and anterior glaucoma.

2. Vascular Pressure. In the glaucomatous eye before operation the ocular tension was 60 mm. of mercury; in the enucleated eye the tension was 20 mm.

It is evident that this great difference, between 60 and 20 mm., represents the participation of the general circulatory factor in the hypertension, and if one compares this figure with the similar one for the normal eye one cannot but be struck by its importance in glaucoma.

Sclerosis of the vessels was long ago observed in anatomic examinations of glaucomatous eyes and especially as involving the veins.

I also drew attention to this in 1912 in a study of three cases of infantile glaucoma. Bailliart and I, in 1925, spoke of the circulatory factors in glaucoma, and in 1929, I once more insisted on their importance.

These vascular lesions are not specific. They resemble those met with in other ophthalmic diseases without glaucoma. It is, however, interesting to note a certain hypertrophy of the walls of the arteries as if these vessels require reenforcement to combat the pressure syndrome. As to the veins, phlebitic lesions have almost always been described. Lastly, tonoscopy at the disk with Bailliart's method shows that in all glaucomatous eyes the venous pressure is always high and at about the level of the ocular tension. But the pressure is especially increased in the capillaries. This is known thanks not only to the tonoscopic method, but also to the entoptic investigations inaugurated by Fischer and continued by Dieter. Dieter pointed out an important factor. He showed that the pressure in the capillaries of the fovea

remains high even after the ocular tension has been reduced by operation.

In fact, I take it that in glaucoma the initial lesions are above all lesions of the capillaries causing an increase in the permeability of their walls and an increase in the pressure of the blood. A certain hindrance in the venous circulation augments and contributes greatly to a still further rise of the blood pressure, first of all in the capillary network and then in the arteries, which are forced to combat this obstruction. I do not think, however, that the increase of the tissue fluid is due solely to an increase in the pressure in the capillary network, but I believe that the increased quantity of aqueous and the high capillary pressure are two factors which are interrelated. At the Amsterdam Congress I endeavored to show that the causes behind these phenomena might be diverse: toxic, infectious and, above all, nervous.

The nervous element is very important, whether one considers it as a disturbance of the sensory innervation or as a disturbance of the sympathetic system, as believed by Hamburger, Thiel and others. The existence of the nervous influence has been known since the time of Daviel and appears very often in the etiology of the attack of glaucoma. It shows itself likewise in clinical practice under another form, spasm. This spastic state is not often discernible with the ophthalmoscope, but can be demonstrated by certain characteristic campimetric defects. These scotomas, whether they be of the form described by Bjerrum, by Seidel or by Elliott, are angioscotomas, and Traquair, in his book, duly insisted on their nature. It is known also that they are not specific to glaucoma but that they are found in various diseases: in intoxication by quinine (Laddoni), in choroiditis and in nose and sinus infections (Dijentz and Cholina). They occur even in old men affected merely with senile sclerosis of the retina (Salzer). One of the outstanding characteristics of scotomas in glaucomatous patients is their frequent daily modifications in size and shape. It was thought that these changes were due to modifications of the ocular tension, but experience has shown Traquair that their form and their extent are independent of the hardness of the eyeball. As these are angioscotomas, it is normal to believe that the variations are due to spasm.

In thinking of this question some other facts deserve the most serious consideration. Observations have been published on glaucomas in which injections of acetylcholine brought about an increase in the visual field. I have myself observed several such satisfactory results and often use this medication. But no observation is more instructive than that of Gallois. He reported a case of double glaucoma in which a patient who had lost one eye suffered an almost complete reduction of the visual field of his remaining eye. Gallois employed acetylcholine. The ocular tension was moderate when the first injection was made, but

the observer was surprised after a few days to note that the visual field was progressively enlarging, while at the same time the ocular tension was increasing. Thus in spite of a higher tension, function was reestablishing itself and sight was improving. Such observations prove that in glaucoma the diminution of vision is not due to the hardness of the eyeball.

Sclerosis of the vessels and spasm are two influences which are additive, but it is above all the sclerosis which causes the definitive loss of sight. The hypertension may contribute to the optic atrophy, but numerous clinical facts show that it is not the hardness of the eye which alone causes it. First of all, the disk is served by the vessels of Haller's circle which is situated behind the globe. Secondly, the form of the visual fields in glaucoma does not agree with that theory which explains the optic atrophy as being due to the compression of the fibers on the scleral margin of the disk (Traquair). It must be added that in von Graefe's amaurosis with excavation one observes the same cupping, and it is known that cases of glaucoma, such as observed by Elsching, Heilbrun, Stock, Beard and Brown, and Köllner, have evolved toward optic atrophy without any rise in tension. Therefore, degeneration of the nerve fibers is due much less to the pressure of the vitreous than to the lowering of their metabolism which is interfered with by edema, and above all by the prolonged disturbance of the capillary supply. Later on, the sclerosis of the arterioles and the spasm increase the ischemia, the nerve fibers disappear little by little and the unsupported lamina cribosa is easily pushed back by the vitreous, even when that pressure is not unduly elevated.

Axenfield and others have quoted observations in which, after sclerectomy, the excavation of the disk diminished. This fact would not be surprising if the hypertension of the eyeball had been lowered before the optic atrophy was complete. There has even been a case (Bleiker) in which, following a stroke, the atrophic glaucomatous depression was partly filled up by a further increase in the edema, resulting from an increase in the intracranial pressure, but there still remained in the center of the disk a deep funnel-shaped depression.

CAPILLARY PERMEABILITY IN GLAUCOMA

Among the theories which have been advanced to explain glaucoma, the physicochemic theory which endeavors to account for the pathology by the turgidity of the vitreous has met with great favor.

This theory which general pathology has already made use of for the pathogenesis of edemas, a theory which takes into account the variations of the p_H or of the iso-electric point, offers perhaps a partial explanation, but such physicochemic changes can be considered as only a secondary factor in the vascular disturbance.

In fact, in glaucoma the anatomic element first affected is the capillary itself. Abnormal changes occur in these vessels which both alter the permeability of the endothelial walls and obstruct their lumina. These pathologic changes cause most of the clinical signs.

The modification in the permeability of the walls must be considered as being twofold in its effect: a modification in the exchange in the direction blood to aqueous, i. e., an increase in the production of the tissue fluid, and a modification in the exchange in the direction aqueous to blood, i. e., a diminution in the reabsorption of the fluid. In the normal physiologic state the hydrodynamic pressure of the blood being inferior in the arterial end of the capillaries to the colloidal-osmotic pressure, dialyzation takes place according to a normal qualitative and quantitative relationship. When the hydrodynamic pressure diminishes, owing to the extreme division of the capillary network, the osmotic pressure of proteins becomes higher and attracts the fluid toward the blood; this accounts for the reabsorption which takes place in the venous end of the capillaries.

In order to explain the increase of the aqueous one might therefore suppose the existence of a modification in the osmotic pressure of colloids, but researches made along this line have shown that in glaucomatous patients this pressure of colloids does not differ from that of normal persons. One must suppose, then, that the increase in the permeability of the endothelial cells is due to another cause.

On the other hand, experimental medicine has shown that an increase in the permeability can be produced by the use of toxic substances, for instance, by injecting into the veins snake venom (Gunn and Frazer) or histamine (Dale and Richard). This is why Duke-Elder stated that glaucoma might be explained by some intoxication of the histamine type, that is, by substances of this type normally existing in the body.

The endothelial cells which preside over the dialyzation can be injured in various ways. It seems logical to suppose that a toxemia might alter their normal function. The function of the endothelium may also be indirectly altered by nervous disturbances. Indeed, section or stimulation of the cervical sympathetic, and stimulation or section of the fifth nerve modify the chemical composition of the aqueous humor. Now, in glaucoma, the nervous factor appears to play a very important part (increase of sympathetic tonus [Hamburger], diminution of sympathetic tonus [Thiel]). Krogh and his pupils have indeed demonstrated that the capillary vessels are affected by three principal influences: the hydrodynamic factor, the nervous factor and the humoral factor.

The influence of the endocrine glands in glaucoma has been much discussed, but knowledge on this subject is very rudimentary, not only

on account of the interdependence of the glands themselves, but also on account of their relations with the nervous system (which has been designated under the name of neuro-endocrine complex).

After all, one must acknowledge that in glaucoma one is more familiar with the hydrodynamic factor than with either of the other two. There was even a time when, having observed that many patients were affected by a high systemic blood pressure, it was thought that this factor alone was capable of inducing glaucoma. Clinical practice has shown that this is not always the case. Certain glaucomatous patients have normal systemic pressures.

But it must be remembered that systemic pressure and local blood pressure are two different things. The researches which I have made with Bailliart show that, whether the patient suffering from glaucoma has a high or low systemic pressure, in every case there is a high local blood pressure. The rise in the capillary blood pressure reacts on the arterial pressure which shows a great difference between the maximum and minimum (differential pressure), but the venous pressure is especially affected.

This increase of the capillary pressure may be due to two causes. There may be some hindrance in the venous circulation and also a diminution in the caliber of the capillaries. It is likely that it is a combination of the influence of these two factors which causes the high local pressure. It is known from Thiel's experiments with fluorescein that in glaucoma this dye appears more rapidly in the anterior chamber than it does when instilled in the normal eye. This again indicates an increase in the permeability of the capillaries. Besides, Dieter's entoptic observations have shown that the capillary pressure of the macula is always very high in glaucoma. One might think that the mechanism of the so-called peripheric hearts (contractions of the arterioles) developed the increased arterial and capillary pressure in order to combat the ocular hypertension. But Dieter has pointed out that the pressure remains high even when the ocular tension is reduced by sclerectomy. It is therefore impossible to attribute the increase of the capillary pressure to the ocular hypertension, and one is led to think that it is the first factor, that is, the increased permeability of capillary walls, which reacts on the second.

Under ordinary circumstances, as described by Langley (1911) and Krogh (1919) and the beautiful observations of Richards in Philadelphia on the glomerular circulation, all of the capillaries of a limited region are not filled with blood; some of them are empty and appear contracted. The increase in the capillary venous pressure can therefore be explained if one thinks of these empty capillaries being brought into play and the whole capillary network functioning.

Anatomic examinations and also the frequency of glaucoma in certain diseases of the vessels, such as pulsating exophthalmos and nevus of the face, give additional weight to this argument. It is also known that compression or massage of a glaucomatous eye lowers the tension very slightly and only for a short time, while this temporary amelioration is followed by a very strong reactive phase (Dieter, Magitot, 1931) which indicates a high blood pressure and some disturbance in the reabsorption of the ocular fluids.

Yet this mechanism in the circulatory picture must play only a predisposing rôle. All forms of exophthalmos, all nevi of the eyelids, all thrombosis of the central veins are not accompanied by glaucoma. Thus one must conclude that the blood pressure in the capillaries may be raised by another factor. Very strong presumptive evidence permits the suggestion with considerable certainty that this other factor which one must seek is to be found in the nervous system. I would point to the nervous control of the blood vascular system. A derangement of this vasomotor mechanism controlling the ocular circulatory bed might be brought about by various toxic or infectious agents acting either directly or indirectly on the nervous system. It is even possible that this derangement or increased sensitivity might be a disease of the vago-sympathetic system itself. On the other hand, one might consider that an indirect influence through some imbalance in the internal secretions was brought into play.

Let me consider a little further the theory of a possible nervous factor entering prominently into the etiology of glaucoma and accounting for many of the phenomena which one observers but cannot logically attribute to any other cause. In the initial period of glaucoma, before the increase of blood pressure in the capillaries is complicated by sclerosis and a permanent contraction of the vessels which ends with atrophy of the tissues, therapeutic measures often have a favorable effect. So long as the circulatory disturbance is not complicated by these pathologic changes, various measures may bring about equally good results. Since the time when von Graefe discovered the favorable action of iridectomy and Laqueur made use of physostigmine, ophthalmologists have had at their disposal both this surgical and this medical means of treating the disease.

Surgical methods have multiplied, but contrary to what takes place, for instance, in an operation for appendicitis, in which the cure is obtained by the removal of the affected organ, in glaucoma technics are employed which do not attack the disease itself but endeavor to combat the hypertension in the eye. This hypertension is indeed the symptom which has been classically confused with the disease. What is still more striking in surgical treatment is the good results which appear from very diverse operative technic. One cannot doubt the sincerity of the

advocate of each of these different methods. In the hands of some surgeons simple iridectomy may be quite as efficacious as sclerectomy, and experience shows that between two sclerectomies the one which creates a cicatrix in the form of the classic filtrating button often has a less favorable result than the one which produces a flat scar.

In reality I am convinced that all antiglaucomatous operations may be either bad or good regardless of technic, because they act only by nervous shock and affect the cause of the disease only in an indirect manner. The best result is no doubt produced by that operation which induces the strongest nervous shock. As experience has shown, the most lauded technics are sometimes inefficacious and even cause an aggravation instead of an amelioration of symptoms.

Turning to medical therapy, one finds a similar state of affairs. Physostigmine, pilocarpine and epinephrine all have a favorable action for two reasons: These drugs reduce the permeability of the endothelial cells and regulate the blood stream. After instillation of physostigmine drops, von Hess, who made entoptic studies on himself, was able to observe modifications of the perifoveal capillary circulation, while Michaïl and Vancea, observing the vessels of the conjunctiva through the biomicroscope, drew attention to the disturbance of the blood stream. Sven Larsson has made the same observations on the vessels of the iris. But to this circulatory factor is certainly added an action on the permeability of the endothelial cells. Gala's experiment gives evidence in this direction. If one injects into the veins of an animal a solution of sodium iodide this substance normally appears very rapidly in the ocular fluid, but if beforehand one instils into the conjunctival sac drops of physostigmine, one finds no trace of sodium iodide in the aqueous humor.

Epinephrine, which is nowadays often employed, possesses an action, if not of the same order, in any case quite comparable to that of physostigmine. From Wessely's experiments one knows that, thanks to a subconjunctival injection of an epinephrine solution, the appearance of the plasmoidal humor following a corneal puncture can be avoided. If on the other hand one studies the action on the glaucomatous eye, one observes that its effect on the ocular tension does not manifest itself immediately but only after several hours have elapsed, and often as late as the second day after its instillation.

It seems quite natural to wonder if these various substances have not a common point of attack. This is rather unlikely, for pharmacology shows that these drugs are amphoteric with regard to the sympathetic nervous system and that they act both on the sympathetic and on the parasympathetic. Perhaps the question is too complex to be solved at

present. However, the probabilities remain that one common point of attack is the endothelial cell itself while simultaneously an action on the nerves which govern cell metabolism is brought about. Vasoconstriction intervenes, it is true, yet the experiments of von Hofe indicate that the vasomotor mechanism is not exclusively at fault. Glaucomas, whether they be primary or secondary, have a relationship which indicates that if their causes differ, the mechanism which is involved is the same in both categories.

To conclude briefly, let me say that there comes a time, even after having very closely followed up a question and gone so far as to renounce old settled principles, when we must stop; our knowledge fails us. In such a complex question as glaucoma certain points remain obscure even when we have done our best to use all the knowledge at our disposal. Doubtless the scientific progress of tomorrow will supply new light and clear up the whole picture for us. One of the chief objects of my paper has been to remind myself and my colleagues how necessary it is that we ophthalmologists extend our horizons. We must go beyond the rigid limits of the territory of our specialty and bring to bear on the problems of such vital interest to us all the knowledge that the basic sciences of general medicine are continuously disclosing. We must remember to look for similar happenings in other regions of the body.

Glaucoma is not a disease peculiar to the eye. Hypertension in the semicircular canals occurs in certain labyrinth diseases. But one must generalize still further and consider glaucoma as a syndrome which is found also in intracranial hypertensions. When I speak of intracranial hypertensions I mean especially those which are associated with tumors of the brain. These are secondary cerebral glaucomas comparable to those secondary glaucomas known in the eye. One meets also with intracranial hypertensions in the course of meningitis and encephalitis. These resemble the glaucomas secondary to choroiditis and iridocyclitis. Lastly, in the condition which is designated by the term serous meningitis, a meningitis in which the spinal fluid is chemically normal, the disease evolves in a manner which reminds one strongly of simple chronic glaucoma. Other analogies could be found, but it is unnecessary to carry my point further; these comparisons suffice to show that the eye is but a department of the complete human organism and that ophthalmologists must turn toward general pathology in order to solve the problems which they encounter.

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THE BLUE ARCS OF THE RETINA*

BENJAMIN FRIEDMAN, M.D.
NEW YORK

As early as 1825, Purkinje [1] called attention to a phenomenon that has become more intriguing as more facts have been added concerning it. If an observer in a dark room, preferably with one eye closed, looks a little to the temporal side of a circle or band of light, he will see, in addition to the source of light (the primary stimulus), two curved bands of bright blue beginning at the stimulating light and arching across the visual field to the region of the blind spot. At times the enclosed elliptical area is also filled with a faint blue haze.

Several subsequent workers (Zeeman,[2] Tscherning [3] and Hubbard [4]) independently reported the same discovery, being incognizant of Purkinje's original paper because of its inaccessibility.

The cause of the phenomenon has been explained in varying ways. While Purkinje recognized a relationship to the anatomic structure of the retina, he could not suggest a plausible hypothesis. Charpentier [5] believed that light was reflected from the temporal slope of the fovea along the course of the nerve fibers, illuminating them and thereby causing the underlying receptors to be stimulated. However, the blue arcs are not visible when the fovea alone is stimulated, and therefore this explanation cannot be accepted. Ten Siethoff [6] vaguely implied the phenomenon to be an "entoptic complementary after-image." The meaning of this is not quite clear. He believed the blue color to be due to absorption of blue light by the macula. Amberson,[7] in criticizing this view, stated that "inasmuch as the effects appear almost, if not quite coincidently with the viewing of the primary stimulus, and disappear at once with its removal, the time relations hardly indicate an after-image. Nor does the fact that the secondary effects extend far

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* From the Knapp Memorial Eye Hospital.

1. Purkinje: Opera Omnia, 1825.
2. Zeeman: Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **6**:233, 1894.
3. Tscherning: Klin. Monatsbl. f. Augenh. **36**:233, 1898.
4. Hubbard: Psychol. Bull. **7**:196, 1910.
5. Charpentier, A.: Compt. rend. Soc. de biol. **8**:765, 1885.
6. ten Siethoff, F. G. A.: Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **14**: 375, 1894.
7. Amberson, W. R.: Secondary Excitation in the Retina, Am. J. Physiol. **69**:354 (July) 1924.

beyond the limits of the limits of the yellow pigmentation in the macular region seem to justify the second conclusion." (The selective absorption of the macula as the cause.)

Working independently, Hans Gertz,[8] Otto Gertz[9] and Hubbard offered a different explanation, with which Troland,[10] in a later paper, and Amberson and Davis,[11] still later, agreed. They suggested that the electrical currents known to accompany the passage of an impulse along a nerve, starting in the stimulated area and traveling in the fibers of the optic nerve, stimulate secondarily the adjacent retinal elements and thereby produce an effect which is projected in the corresponding portion of the visual field.

In 1914, Druault[12] opposed this theory, and offered another explanation. He believed that the excited nerve fibers are actually luminous from physical light which they emit, and that the underlying receptors furnish the arcs through this stimulus. Six years later, Ladd-Franklin,[13] unaware of Druault's theory, contributed a similar idea of bioluminescence, and coined the term "blue arcs," which is now employed.

Roaf[14] expressed the belief that the arcs are due to "halation," a phenomenon obtained by looking at a bright light: One then sees a surrounding halo of the same color as the light itself. He explained the eccentricity of the arcs as being the result of the nasal location of the visual axis in relation to the fovea.

The arcs may be elicited by a stimulus of almost any color. Red seems to be the best color for bringing them out, possibly because there is less scattering at this wavelength, but green and yellow produce them nearly as well. In a dark room one may see them with a white light, but it is difficult to observe them with a stimulus of violet light, probably because the fluorescence and scattering within the eyeball interfere with their recognition. Hubbard found the "brushes," as he called them, to be faint at the extreme red, brightest at the orange-red and nearly as bright at the yellow divisions of the spectrum. The shape of the stimulus generally employed is rectangular, but any formation will

8. Gertz, Hans: Skandinav. Arch. f. Physiol. **19**:381, 1907.

9. Gertz, Otto: Hospitalstid. **7**:1197, 1914.

10. Troland: An Entoptic Phenomenon Demonstrating the Optic Impulse, Psychol. Bull. **17**:55, 1920.

11. Davis: Science **67**:69, 1928.

12. Druault: Sur un phénomène entoptique en rapport avec le trajet des fibres optiques dans la retine (phénomène des arcs), J. de physiol. et de path. gén. **16**: 649, 1914.

13. Ladd-Franklin, Christine: Science **65**:239, 1927; **67**:162, 1928; Compt. rend. Soc. de biol. **185**:584, 1927; J. Optic. Soc., America **16**:333, 1928.

14. Roaf: Is the So-Called Excitation of the Retina Due to Stimulation of the Rods? J. Physiol **69**:1, 1930.

yield the arcs. I can readily see them late at night when, looking across the street, I focus on a solitary brightly lighted window. The colored traffic signals against a dark background may serve as stimuli. One may see the arcs streaming from the border of the full moon; indeed, once having become familiar with the phenomenon, one encounters it under the greatest variety of unexpected circumstances.

Hubbard, using a circular source of light, made an interesting observation. When he fixed his line of sight to the temporal side of the circle he saw both arcs converging to the blind spot; when he fixated on the upper edge, he saw only the lower arc; fixation on the lower edge produced only the upper arc, and fixation on the nasal edge produced a triangular blue light the base of which lay at the stimulating circle and tapered to its apex at the blind spot. Amberson called the latter effect the "blue spike," and he showed that it lies along the axis joining the macula and the blind spot. Snell so arranged his stimulus in the transition zone that the arcs and the spike were alternately observed, proving that they are but different manifestations of the same phenomenon.

Fig. 1.—*X* makes the point of fixation. After Hubbard.

Amberson has carefully worked out the areas of the visual field where stimulation is effective in producing the blue arcs and the spike. He used a larger and smaller stimulus, and mapped out a larger and smaller corresponding "effective" field. Snell [15] found a larger field than Amberson (15 degrees nasally), but he did not state the size of his stimulus. The fields for the arcs lie to the nasal side of the point of fixation, that of the spike to the temporal side. Snell found the latter to be in a narrow ring concentric with the fovea. The actual anatomic areas of the retina involved, of course, lie on opposite sides of the fovea as compared to their fields. Elevation or depression of the stimulus in relation to the point of fixation causes a decrease in intensity and the ultimate disappearance of the lower or upper arcs. Both investigators used stationary stimuli and movable points of fixation of phosphorescent material.

Amberson called attention to the central area of "blindness" for the arcs. His measurements show an average width of 1 degree 58

15. Snell, P. A.: The Entoptic Phenomenon of the Blue Arcs, Arch. Ophth. **1**:475 (April) 1929.

minutes, which corresponds fairly well with the width of the rod-free area of the retina. Quoting Parsons, he stated that the rod-free area is about 3 degrees wide at its maximal variation; the fovea subtends about 1 degree. He offered this area of "blindness" as a physiologic test for the rod-free portion of the retina. If this argument is acceptable, the rod-free area would lie eccentrically, chiefly to the nasal side of the fovea, and not to the temporal side, as he suggested. He emphasized the fact that the phenomena cannot be elicited by a stimulus falling in the field that corresponds to the rod-free area, and he drew the conclusion that the arcs are rod rather than cone phenomena so far as the stimulus must fall on a region where rods are present in

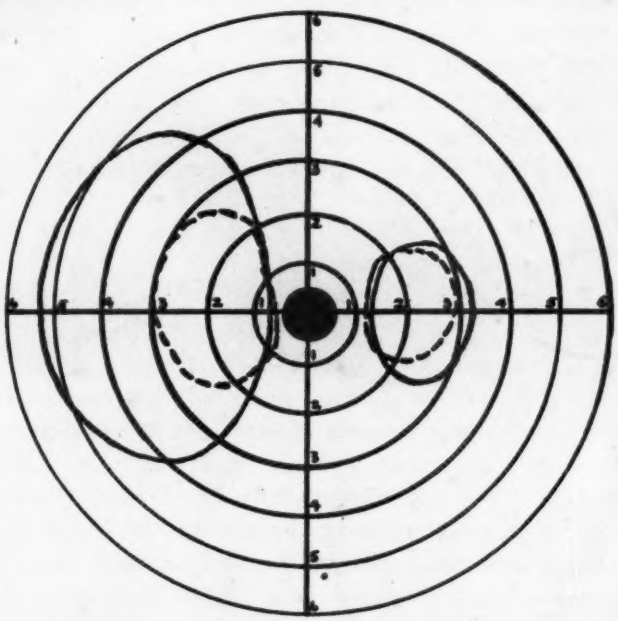

Fig. 2.—"The retinal areas (projected) within which the primary stimulus is effective in producing the secondary phenomena. The central black area represents the size and position of the fovea. The blue arcs are produced when a vertical stimulus field excites the areas to the left; the blue spike when a horizontal field excites the areas to the right. Full lines indicates areas as determined by large stimulus fields; broken lines indicate areas as determined by small stimulus fields. Concentric circles are drawn at intervals of one degree of visual angle" (after Amberson).

order to evoke the arcs. This is applicable to the primary stimulus; there are indications that a secondary system exists which may be related to the distribution of the cones.

Thus, a study of the accompanying table from Snell suggests a direct relationship between the intensity and the thickness of the arcs and the concentration of the cones.

The blue color of the arcs also speaks for a relationship with the cones, the recognition of color being, so far as is known, a property of the cones exclusively. I shall have occasion later to modify this statement.

Judd,[16] by a different method, has attempted to find out whether the rods or the cones are the recipients of the primary stimulus. Using light stimuli of varying wavelengths, he determined the least retinal illumination necessary to evoke the arcs. His reasoning was that if the rods are the elements that stimulate the activity of the nerves resulting in the blue arcs, the strength of the illumination needed will be higher for wavelengths in the red end of the spectrum than for those in the green, because the rods are relatively insensitive to the longer wavelengths. If the cones are the mediators in the mechanism of the blue arcs, the threshold of illumination (measured in photons) will be the

Variation in the Size and Character of the Arcs with a Change in the Region of Primary Stimulation

Distance of Fixation Point from Slit, Mm.	Distance Between Arcs at Maximum, Mm.	Distance of Normal to Curve at Maximum from Fixation Point, Mm.	Character of Arcs
5	9	20–25	Spike; bright edges filled with blue haze
10	17	22–25	Distinct arc; slight haze within
15	25	23–25	Distinct arc; slight haze within
20	33	25	Arcs bright; haze along inner edge
25	42	25	Arcs bright; sharp edges
30	51	26	Arcs less bright; thinner
35	60	26	Arcs quite thin; less bright
40	68	..	Arcs fine lines; faint
45	..	..	Arcs merely suggested; very indefinite
50	..	..	No arcs

same no matter what the wavelength. Analyzing the shape of his "curve of least retinal illumination against wave length," he found a rise in the illumination values for wavelengths above 500 millimicrons (the red portion), indicating an involvement of the rods. In addition, he found certain resemblances between the blue arcs and the Purkinje phase of the after-image, which is conceded to be a reaction of the rods.

It is universally agreed that the arch of the arcs follows the pattern of the nerve fibers between the paramacular region and the disk. This may be verified by plotting on the tangent screen. The blue spike is inclined from the horizontal when the papillofoveal line is so inclined. I find the direction of the arcs and of the spike to alter when the head is turned to the side; they always follow the inclination of the papillo-

16. Judd: Least Retinal Illumination by Spectral Light Required to Evoke the "Blue Arcs of the Retina," Bureau of Standards, Research Paper no. 43, J. Research, vol. 2, Feb., 1929.

foveal line. Karrer and Stevens[17] found that "appropriate pressure" on the eyeball could alter the size and shape of the arcs.

Snell, from experiments with primary stimuli of different colors, concluded that the color of the arcs is always blue, and not reddish-blue or purplish, as has been suggested. He found that the reddish tinge occurred only when using a stimulus of red; it could be eliminated by reducing the light of the stimulus. It seems to me logical to interpret this as an effect of scattering of the red; its disappearance with the decrease in intensity is to be expected.

In 1920, Troland[18] found that the intensity of the arcs was independent of the stimulus, the phenomenon thus concurring with the "all or none law." Ellis,[19] on the other hand, found that within limits the intensity of the arcs does vary with the intensity of the stimuli, apparently defying the "all or none law."

Keith Lucas and Adrian have shown that submaximal contractions of muscle tissue, which vary according to the strength of the stimuli, are due to the failure of the stimuli to excite all of the muscle fibers, but that the fibers that do respond do so to their fullest capacity, following the "all or none law."[20] Adrian and his co-workers[21] have shown that this condition also exists in motor and in sensory nerves, and most probably in sensory nerves from visual receptors. Snell offers a similar explanation for his findings, which showed a variation of intensity of the arcs to graded stimuli above the threshold and below a certain maximum. With intensities above this maximum, the intensity of the arcs did not increase. In the optic nerve the frequency but not the size of the action current is the variable, and since the intensity of the arcs is proportional to the variations in frequency, the "all or none law" is not violated.

Amberson has recorded a most interesting observation on a subject who had a degenerative lesion of the retina lying between the macula and the optic disk. The blue arcs could be seen extending

17. Karrer, E., and Stevens, H. C.: Visual Perception in Motion, Am. J. Physiol. **94**:611, 1930.

18. Troland: The "All or None Law" in Visual Response, J. Optic Soc., America **4**:160, 1920.

19. Ellis, F. W.: Secondary Excitation of the Retina and the Variation of the Intensity of the Resulting Sensations, Am. J. Physiol. **82**:290, 1927.

20. Galfan, recently working with isolated muscle fibers in the retrolingual membrane of the frog, found evidence of graded response, in apparent violation of the "all or none law" (Am. J. Physiol. **93**:1, 1930).

21. Adrian, E. D., and Zotterman, Y.: The Impulses Produced by Sensory Nerve Endings: I, J. Physiol. **61**:49, 1926; II. The Response of a Single End Organ, ibid. **61**:151, 1926; III. Impulses Set Up by Touch and Pressure, ibid. **61**:465, 1926. Adrian, E. D., and Matthews, R.: The Action of Light on the Eye: Discharge in the Optic Nerve, J. Physiol. **63**:378, 1927.

to but not through the scotoma produced by the lesion. Amberson concluded that the blue arcs depend on the integrity of the retinal elements between the macula and the blind spot; they cannot be cortical phenomena because the arcs were present as usual in the healthy eye. The lesion produced a much larger scotoma for blue, but curiously enough, in this blue-blind region a segment of the blue arcs was clearly visible. This patently suggests two different mechanisms in the perception of blue. Ladd-Franklin offered an explanation of this paradox. She assumed that owing to the trauma there was a dispersion of yellow pigment in the retina, and that the yellow pigment absorbed the blue rays from the test object and therefore a scotoma for blue resulted. The nerve fiber layer, however, in one certain area was not covered by the yellow pigment, so that at this point the visible radiation could

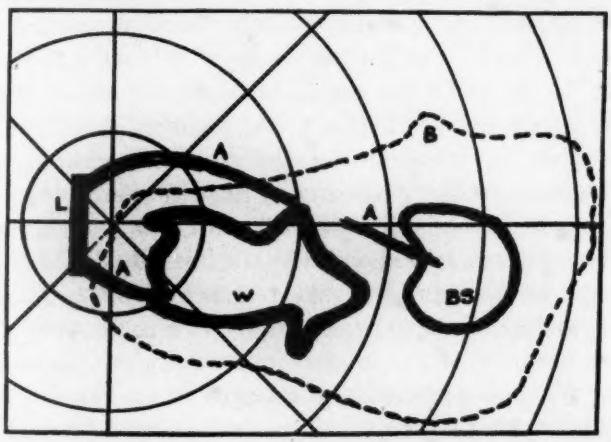

Fig. 3.—*L* indicates the source of light; *A*, blue arcs; *W*, scotoma for white; *BS*, blind spot; *B*, scotoma for blue. Segments of the blue arcs are visible in the region where there is a scotoma for blue. (After Amberson, modified.)

reach the receptor cells unobstructed. While Ladd-Franklin's idea is ingenious, there is little of actual fact with which one may support it; the deposition of yellow pigment is by no means a conspicuous element in the pathology of traumatic lesions of the retina; furthermore, Amberson failed to state whether in the actual case under discussion there was a noticeable amount of yellow pigment in the injured area.

Statements concerning the effects of dark adaptation on the intensity of the arcs are conflicting. From my observations and from those of Judd, I am inclined to disbelieve that dark adaptation has a weakening effect on the arcs, as has been stated. A possible factor of error in all experimentation on dark adaptation is the disturbing and antagonistic effect of the stimulus itself, repeated at intervals, on the state of dark adaptation. Subliminal stimuli are the least prone to such criti-

cism, but the control of the intensities of the stimuli is not mentioned by any investigator working on the relationship of dark adaptation to the intensity of the blue arcs.

Snell, studying the duration of the arcs, found the average time to be about eight-tenths second. It could be prolonged to eight seconds or more by fixing the eye on a moving point and thereby causing the stimulus to fall on fresh receptors continuously. The duration of the arcs could be lessened by allowing only a short interval (one second) between the disappearance of the arcs and the appearance of the next stimulus; in other words, the arcs that followed a stimulus to a partially recovered retina were of shorter duration than those that followed stimulation of a completely recovered retina.

Troland and Snell expressed the belief that the fading of the arcs within one second is due to the ineffectiveness of the retinal action currents as stimuli, for the currents quickly reach a constant maximum, and then, according to the law of Dubois-Réymond, lose their stimulating value, falling below the threshold required for the arcs. Amberson, on the other hand, expressed the opinion that fatigue of the secondary system is responsible for the rapid disappearance.

The mechanism of the phenomenon has provoked much discussion. Druault and Ladd-Franklin defended the idea of bioluminescence of the stimulated nerve fibers. Judd agreed with this. Ladd-Franklin argued that a great many objects of varied structure are sufficiently self-luminous to "take their own photographs." Keenan [22] confirmed this assertion, but found that the interposition of a glass plate between the material and the film prevents any changes in the film; a semiporous obstacle, such as blotting paper, does not. He expressed the belief that the cause was the quasi-light action of hydrogen peroxide, since hydrogen peroxide will produce identical changes on the film. Furthermore, the changes are facilitated under conditions which are conducive to the formation of hydrogen peroxide, and retarded under conditions which are inimical to the formation of hydrogen peroxide. Ellis,[23] working in the dark on stimulated frogs' retinas, was unable to note any luminescence. In weighing this evidence, one must concede that the luminescence might be too small to be detected by the observer's eye and yet be sufficiently bright to be seen by the rods or cones at a distance of a fraction of a millimeter. Also, that the light might be of the nature of ultraviolet light and so become absorbed by the cornea and humors of the observing eye, whereas in the eye of the subject these filters are not to be considered. Ladd-Franklin stated that she saw a colored after-image of the blue arcs—a dark olive color. Troland

22. Keenan: Chem. Rev. **3**:95, 1926.

23. Ellis, F. W.: The Nature of the Stimulus which Excites the Blue Arcs of the Retina and Related Phenomena, Am. J. Physiol. **84**:485, 1928.

and Snell observed a distinct negative after-image. Others have denied that an after-image is formed. I have been able to see distinctly a colorless positive after-image. H. Gertz reported an after-image of a weak yellow color. Ladd-Franklin contended that only physical light can produce a chromatic after-image; that mechanical or electrical stimulation cannot do it.

A more impressive plea has been made for the theory of secondary electrical stimulation. The action current of stimulated nerve is a long known discovery. Beginning with Dubois-Réymond in 1819, a large number of investigators have demonstrated these currents in the retina by means of a nonpolarizable electrode on the cornea and another on the optic nerve or retina. These currents are distinct from the currents of injury. With the incidence of light, the retina becomes electronegative to the cornea; that is, the current flows outward along the line of sight from the fundus to the cornea. In 1925, Chaffee, Bovie and Hampson,[24] by an intricate and delicate method, found that this current contains a number of summits or maxima, which might be separated into four components, the first two of which they showed to be separate reactions of the rods and cones, bearing out the duplicity theory of von Kries concerning color vision. The curves follow quantitatively the established laws of retinal reaction to light. These findings may be accepted as true indicators of a series of electrical variations in the retina attending its stimulation by light, and it requires no great stretch of the imagination to concede the possibility of these currents secondarily stimulating the retina.

Which of the elements are secondarily excited? The contiguous nerve fibers cannot be responsible, for their projected location would depend on the position of their cell receptors, and these would not be in the form of the blue arcs. The secondary effects, then, must be on the underlying ganglion cells, the bipolar cells or the layer of rods and cones. It is reasonable to assume that the ganglion cells being directly adjacent to the nerve fibers, would be the most strongly affected by the action currents. The rods and cones, as Amberson pointed out, are distantly situated from the electrical fields (from 0.3 to 0.4 mm.), and would probably be little influenced. It is stated that the rods cannot be solely responsible, for they respond only with a white or a gray sensation to a stimulus of any color. Even the perfect functioning of the cones, as is seen in the pathologic case previously described, is apparently not needed to perceive the blue arcs, for the latter were seen in an area where the cone cells were unable to distinguish a blue test object or the blue of a prismatic band. There are no special reasons to believe that the bipolar cells are the intermediaries. I have found

24. Chaffee, Bovie and Hampson: J. Optic Soc., America **7**:1, 1923.

that under some conditions the arcs leave an after-image. Since they leave an after-image, the photochemical substance of the retina must be involved. To disintegrate the photochemical substance one must hypothecate a flow of impulse in a backward direction from ganglion cells to rods or cones; such a backward flow is not inconsistent with experiments on the directional flow of nerve currents. There is a weak link, however, in this conception; it predicates a flow of current during the period of visibility of the arcs at once in a backward direction from ganglion cells to the rods and cones, and in a forward direction from the same rods and cones through the same set of ganglion cells toward the cortex.

An examination of patients with suitably located medullated nerve fibers for possible breaks in the continuity of the arcs would not, on theoretical grounds, help decide whether the arcs are the result of visible radiation or of secondary electrical stimulation. This is because the medullated fibers must be considered as being insulated both optically and electrically.

To summarize the mechanism, then, it is found that a light stimulus of any color, falling on the temporal and the nasal paramacular regions, will produce the arcs and the spike respectively. Since stimulation of the cones alone by the primary stimulus will not evoke the arcs, it is assumed that the initial impulses must be set up in fibers arising from the rods. The electrical discharges or the visible radiation along these fibers affects the underlying retinal elements, either the bipolar cells connected with the rods or cones or possibly both, or else the rod-cone layer directly. If one accepts the theory of an electrical discharge, one assumes a transfer of stimulus from nerve fiber to adjacent cell without benefit of synapse. If one accepts the theory of visible radiation, one admits a condition which has no parallel in nerve tissue. There is too great an insufficiency of fact and too much of contradictory evidence to render judgment in favor of either side at this stage. No one has explained the constancy of the blue color.

The following observations may have a bearing on the ultimate solution of the problem, particularly on the baffling sameness of the blue color. A similar blue may be obtained by pressing lightly with the hand over the closed eye in a dark room; often the pressure is not even needed. A blue circular band advances from the periphery toward the macula, and, having covered it, disappears just as another blue wave closes in. If one presses harder, a greenish or a reddish tinge may obtain, but these colors are drab and vague, and are not saturated like the blue. It thus appears that the receptors for blue are the most easily stimulated. Those for other colors require more than the crude stimulus of pressure to call forth their respective sensa-

tions vividly; they require actual light. König [25] and Tscherning [26] supported with other data the greater sensitivity of the blue receptors. I believe that the electrical currents from the fibers associated with the arcs act as undifferentiated stimuli; the expected response, if the foregoing interpretation is correct, would be blue because the blue receptors are the easiest to affect; it would be blue no matter what the color of the original stimulus, for the electrical current is qualitatively the same for all colors, as Chaffee, Bovie and Hampson have shown.

The color of the arcs is a function of the intensity of the stimulus employed and of the retinal region stimulated. With weak stimuli the color is gray; with stronger stimuli, the color is blue. Observing the arcs and at the same time pressing on the eyeball, I first see the arcs as the usual blue bows; they gradually fade to a gray and then to a colorless pattern which cannot even be called gray. On further pressure, they disappear altogether. It requires several seconds after pressure is released for the arcs again to appear bright blue; they must pass through a reversed sequence—colorless, then gray and finally blue. The pressure disturbs the mechanism for the recognition of color.

The blue color must result from a reaction of the photochemical substance, and if the theory of electrical excitation is the correct one, the impulse responsible must flow from ganglion cell to rod-cone layer in a direction opposite to the usual one.

Are the cones necessarily involved, as the blue color suggests? I have previously stated that only the cones are concerned in color vision. However, for a long time it has been held that the rods also, under proper conditions, are capable of discriminating blue. Helmholtz [27] stated that the lingering blue color of the evening sky and the blue appearance of a snow-covered landscape by moonlight are discerned by the rods after the cones have lost their ability to distinguish color and vision is rendered scotopic. Siven [28] defended this point of view. According to him, the dim spectrum is not gray, as ordinarily stated, but is definitely bluish. He referred to the relatively increased visibility of blue at certain times which is illustrated by Helmholtz' examples. This is the phenomenon of Purkinje [29] in decreasing

25. König, A.: Ueber dem menschlichen Sehpurpur und seine Bedeuting für das Sehen, Sitzungsb. d. preuss Akad. d. Wissenschaft., Phys.-math., 1894, p. 577.

26. Tscherning: Ann. d'ocul. **159**:632, 1922.

27. Helmholtz, H.: Handbuch der physiologischen Optik, Leipzig, L. Voss, 1867, vols. 1 and 2.

28. Siven: The Rods as Color Perceptive Organs, Arch. Ophth. **42**:156, 1913.

29. The Purkinje phenomenon is based on the fact that with a spectrum of decreasing intensity, the region of maximum luminosity shifts from the yellow to the green portion of the spectrum, that is, toward the shorter wavelengths. Reds then become darker, and blues relatively brighter. The Purkinje phenomenon, if present in the macula, is negligible there, and so there is a relative central scotoma for blue in twilight vision.

intensities blue is not seen by the macula, but is seen by the periphery. He linked the disturbed recognition of blue with disturbances in the rods. Thus, in santonin poisoning he found that the eye was violet-blind; therefore, objects appeared in the complementary color—yellowish green (white minus violet). But the central 8 to 10 degrees did not show this anomaly; he believed the visual purple in the rods to be affected, a contention supported by Filehne's [30] investigations on frogs. In patients with icterus, also, yellow vision was absent from the central area. The affinity for visual purple of the bile salts is well known; since the region of the cones was unaffected, and since, moreover, the visual purple has never been satisfactorily demonstrated in the cones, the assumption is that there is here also a rod disturbance. The significant fact is that night-blindness occurs in cases of icterus, and night-blindness (rod-blindness) is usually accompanied by blue-blindness.

The tenet of rod perception of blue is not utterly unreasonable when one considers that in the metamorphosis of color vision the rods were probably the earliest recipients, and the primitive color sensation was probably blue. Observations on insects tend to corroborate this. It is natural to expect the periphery to have been less sensitive to the blue color than the central regions. When the macula was differentiated, the primitive color sense of the rods was pushed into the background, but it may still be demonstrated under the special conditions described. One is now in a position to understand why the blue arcs, if they are solely rod phenomena, appear bluer when the more central fibers are stimulated. It is more difficult, however, to explain why there is such a rapid loss in intensity of the arcs when the periphery, where the rods are most numerous, is stimulated.

30. Filehne: Ueber die Einwirkung des Santonins und des Amylnitrits auf der Sehakt, Arch. f. d. ges. Physiol. **83**:369, 1900.

EXPERIENCES WITH THE GONIN OPERATION*

MARK J. SCHOENBERG, M.D.
NEW YORK

In a previous paper I endeavored to call the attention of the medical profession to the Gonin procedure reputed to give better results than the methods hitherto used. At that time my experience was limited to ten cases. Since then I have operated on, or assisted other colleagues in operations on, sixty more patients. It is my purpose in this paper to report on experiences thus far acquired.

The renewal of interest among ophthalmologists all over the world in the problem of detachments of the retina has brought out a few important facts that may serve as a starting point for more fruitful investigations. Some of these are: (1) In a group of cases the general condition has something to do with the occurrence of detachment of the retina; (2) in a large majority of cases there is a profound change in the colloidal status of the vitreous; (3) a tear is found in the retina in about 75 per cent of the recent cases; (4) the retina shows degenerative changes over more or less extensive areas; (5) the choroid is also the seat of lesions in a great majority of cases; (6) in some instances even the sclera presents some disturbance, such as thinning of its walls, slight bulging and patches of engorgement of the episcleral tissues.

What the meaning of all this may be is for future investigators to decide. At present there is an urgent need for methods whereby one can study more minute changes of the entire vitreous, not only of the quasi-conical portion situated between the lens and the posterior part of the fundus, but also of the entire inner surface of the eyeball (the ora serrata and the ciliary body). Since the number of retinal tears and chorioretinal changes in detachment of the retina increases as one approaches the region of the equator and ora serrata, it is logical to expect to obtain a great deal more information from the study of that important but as yet clinically inaccessible area of the ciliary body, ora serrata and base of the vitreous region.

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* From the Knapp Memorial Eye Hospital.

DIAGNOSTIC DIFFICULTIES

Limited shallow detachments situated at the periphery and small tears very near the ora serrata and located behind a fold are difficult to recognize. In such cases repeated examination with the aid of both direct and indirect ophthalmoscopy, the patient being successively in the erect and the recumbent position, are necessary to reach a satisfactory diagnosis. The pupil must be dilated ad maximum, and since atropine and homatropine do not always give a maximum dilation (cocaine often clouds the corneal epithelium), an epinephrine or ephedrine "pack" or even a subconjunctival injection of a few drops of these solutions is necessary to obtain as large a pupil as possible.

Another diagnostic difficulty is the determination of whether or not one is dealing with a "pseudotear." This occurred in two cases observed. One could see plainly a bulging retina and an irregular red spot which looked much like a tear. When the patient lay down, however, one could make out that in front of the retina, in the vitreous, there was a membranous opacity with a hole in its center. Behind this hole a retinal blood vessel was plainly visible. There was no tear in the retina.

The diagnosis of a subretinal tumor is at times difficult if associated with a retinal detachment. In a patient of Dr. F. Bracken, of New York, there was present a marked elevation of the retina on the temporal side of the macular area. The side walls of this elevation sloped so abruptly and the retina appeared so taut that one could not help but think that a solid mass lay underneath it. After a few days of rest and a diagnostic injection of tuberculin (which produced a marked cutaneous reaction) the tumor-like elevation flattened out entirely, and the retina became detached in the lower part of the fundus. During the following few days the detachment disappeared entirely. Another case began with a very shallow detachment of the retina in the upper portion of the macular area. In the course of a few days it became a retinal bleb about 4 or 5 diopters in elevation, but it cleared up rapidly after a diagnostic injection of tuberculin (which gave a strong cutaneous reaction). After the disappearance of the detachment, choroidal disturbances in the macular area of both eyes could readily be seen.

Finally, of unusual interest is the following case:

A highly myopic woman, about 38 years of age, presented a retinal detachment and a tear of moderate size just behind the ora serrata on the upper temporal side of the fundus. Having been interested in the clinical study of the zone between the limbus corneae and ora serrata, I transilluminated this zone and found a dark round area, about 4 mm. in diameter, extending between the perilimbal dark ring and the ora serrata. In view of the possibility that this dark zone may be due to a tumor, I postponed ignipuncture. I have watched this patient for about fourteen months. During this time the retina became totally detached and the small

round dark zone gradually extended all around the limbus. Today instead of the light ring that is normally found by transillumination between the corona ciliaris and the ora serrata there is a completely dark ring. Whether this is a new growth or some other infiltrative process remains to be seen.

Aphacic eyes present at times great difficulties on account of the existence of remnants of the capsule and a small and bound down pupil. Under such conditions it is almost impossible to study the periphery of the fundus.

A pronounced cloudiness of the cornea, lens or vitreous makes ophthalmoscopy difficult or even impossible.

More information is obtained from the study of the vitreous by slit-lamp examination in addition to ophthalmoscopy than from the latter method alone. The condition of the vitreous is at times far from normal; at other times faint changes are discoverable only after a great deal of patient study. In recent cases some of these changes are in the deep layers in apposition with the retina. Clinically one may find one or several of the following conditions: (*a*) minute dustlike opacities; (*b*) flocculi or threadlike opacities of various sizes: (*c*) membranous opacities (shreds of retina); (*d*) conglomerates of dust or of flocculent masses; (*e*) large globular pigmentary opacities; (*f*) gray masses of exudate; (*g*) blood, in the form of streaks or lamellae infiltrated between the vitreous strata, usually adherent at one of their extremities to the inner wall of the eyeball (the choroid or ciliary body). The slit light thrown into the vitreous reveals many of these details. One of them, not mentioned in literature, is the separation of the vitreous in several strata. This appears as if the light passed successively through media of greater and less density, so that the vitreous seems to consist of grayish and optically empty layers succeeding each other in an anteroposterior direction. Some of these types of opacities may be seen in the subretinal fluid if it is collected at the time of ignipuncture and immediately studied in vitro with the aid of the slit-lamp.

The important point to be emphasized in connection with the study of the vitreous is that the clearer the vitreous and the nearer it is to normal, the better is the prognosis, other things being favorable.

The mapping out of the field of vision in cases in which there is only a shallow and limited detachment of the retina is more successful in reduced illumination and with colored test objects (particularly blue and green).

INDICATIONS AND CONTRAINDICATIONS

As it is my object to report on personal experiences related to the Gonin operation, it is necessary to deal with the question of classification of retinal detachments as far as it is related to this surgical procedure.

Professor Gonin has emphatically insisted that only cases of retinal detachments presenting a hole or a tear in the retina are suitable for his method of treatment. Purely from the point of view of this method, retinal detachments with tears may be classified in the following three groups: (1) operable, (2) nonoperable and (3) doubtful.

The indications that would stamp a case as operable are: (1) a detachment of relatively short duration (at the most, from two to three months); the shorter the age of the detachment, the more definite is the indication; (2) the small number of retinal tears, their small size and their location in an accessible area; (3) a relatively normal looking vitreous, iris and retina.

The operative contraindications are: (1) a detachment of long duration or a total detachment even of short duration; (2) the absence of a tear or the presence of a very large tear, principally at the ora serrata (retinal dialysis); (3) cloudy media interfering with proper ophthalmoscopy; (4) a greatly degenerated vitreous and retina and active iridocyclitis.

Thus far, the number of inoperable cases is large in proportion to the other two groups. As far as the doubtful group is concerned, not only the personal experience but also the temperament of the surgeon are the determining factors in classifying a case in this group. Should one operate in doubtful cases? Yes, if one has the feeling that without the operation the vision will completely deteriorate, and that the operation may benefit the patient.

The recording of the changes in the fundus, the method of localizing on the surface of the eyeball the area corresponding to the tear in the retina, the landmarks in the topographic anatomy of the surface of the eyeball, the various data regarding the measurements of the eyeball and the technic of the Gonin operation have already been elucidated.[1]

Experience has shown how essential it is to keep a complete record not only of the changes in the fundus, but of the condition of the entire eyeball (vitreous, uvea, tension, blood vessels, etc.) as well as of the functional attainments (visual acuity, fields of vision in good and reduced illumination, etc.). The exact localization of the area corresponding to the tear in the retina on the outer surface of the eyeball is still a matter of individual experience. This area is situated at the crossing point of two lines: the meridian and the parallel passing through the retinal tear. Each of these two lines has its own importance. The parallel determines the distance of the area from the limbus

1. Schoenberg, M. J.: The Gonin Operation for Detachments of the Retina, Arch. Ophth. 3:684 (June) 1930.

corneae and the meridian indicates the direction in which the tear is to be found. Of these two, the former is the more difficult to find, and the accuracy of its localization is much facilitated by the examination of the eye with the pupil dilated ad maximum.

To the "landmarks" (location of the long ciliary arteries and nerves, the venae vorticosae, the lines of insertion of the recti and oblique muscles) succinctly mentioned in my earlier paper, one may add the following: 1. The dark ring of the ora serrata. This is plainly visible in blond persons under good transillumination. By directing the light from underneath the lower lid toward the center of the eyeball while the patient looks successively downward, to the right and to the left, one can see clearly the dark narrow ring of the ora serrata. The application of the source of light from the upper lid, while the patient looks upward, may bring into evidence (though with greater difficulty) the lower sector of the ora serrata. Thus one can outline the ora with methylthionine chloride U. S. P. (methylene blue). 2. The lateral margins of the recti muscles. These are to be avoided by the scissors, knife and puncturing cautery; they are provided with more nerve fibers and blood vessels than other portions, and consequently the bleeding is more profuse and the pain more acute.

THE TECHNIC OF THE GONIN OPERATION

The various steps in the Gonin operation have been amply described elsewhere.[1] However, a few comments regarding experiences during the past year may be useful.

1. It is not advisable to use instillations of cocaine as a local anesthetic. This drug injures the corneal epithelium for the time being and makes ophthalmoscopy difficult immediately after the ignipuncture.

2. A strabismus hook introduced underneath the tendon of the proper muscle is of considerable aid in pulling the eye in the direction desired so as to make an area situated far behind the equator accessible to the cautery tip.

3. When possible, the incision in the conjunctiva should be at least 3 or 4 mm. behind the area of ignipuncture. After the incision, the conjunctiva is undermined with a few snips of the scissors, and a lid retractor (Desmarre) is introduced underneath the conjunctiva and Tenon's capsule. In this way a larger area of sclera is exposed and the margin of the lid is protected from accidental contact with the cautery tip. If the area to be punctured happens to be underneath one of the recti muscles, it is permissible to perform a preliminary temporary tenotomy.

4. The lips of the scleral incision often come so closely in apposition that one fails to find the opening. It is perhaps better to make a mark on the sclera with india ink or with the cautery before the Graefe knife is introduced.

5. The cherry red hot tip of the cautery should be introduced about 3 or 4 mm. into the vitreous, directed toward the center of the eyeball and kept in from two to four seconds. If one wishes to cauterize a larger area of the retina and choroid, one can reintroduce the cautery tip bent in the shape of an "L."

6. It is both useful and instructive to examine the fundus immediately before and after the incision of the sclera and after ignipuncture. Since the operation is performed when the patient is in the recumbent position, the final decision as to the exact location of the tear should be made while he is on the operating table. Occasionally the location of the tear shifts during the change from the sitting to the recumbent position. After the escape of the subretinal fluid there is again more or less displacement of the retina and tear, and this should be ascertained by an ophthalmoscopic examination.

7. Finally, after the ignipuncture the ophthalmoscope reveals the extent of the cauterization and also whether the retina has been anchored on the choroid. One can see a dark round area (the hole in the sclera) surrounded by an almost black, more or less complete, narrow ring, outside of which there is a grayish-white zone of partly edematous and partly necrosed retina. The extent of this white area depends on the size of the cautery tip and the length of time that it has been kept in the opening. In other words, the result depends on the amount of heat per second applied to the tissues of the eyeball. At times no vitreous flows out after the scleral puncture. This is usually due to the thickness of the albuminous subretinal fluid.

8. As soon as the sclera has been exposed it should be carefully inspected. Some cases show a slight bulging, and others a thinning of the sclera, with the choroid shining through; in other cases, one finds a marked engorgement of the episcleral blood vessels. Whatever the meaning of these observations may be, it is wise to make a record of them.

9. Though a minor detail, it is nevertheless important to instil salt solution frequently and regularly into the conjunctival sac. The cornea must be kept moist to make ophthalmoscopy possible. It is of paramount importance to record at the conclusion of the operation the principal steps, the unusual observations and the complications encountered.

10. The binocular bandage is to be removed every two days, the lids cleansed and a drop of a 1 per cent solution of atropine instilled (never salve, because it interferes with ophthalmoscopy).

11. Dr. H. Goalwin's formula serves fairly well for the calculation of the axial length of ametropic eyes: The axial length of the emmetropic eyeball being 24.35 mm., to calculate the axial length in ametropic eyes, subtract from 24.35 mm. one-third millimeter for each diopter of hyperopia up to 10; add 0.5 mm. to 24.35 mm. for each diopter of myopia up to the first 10; add 0.6 mm. for each of the next 6 diopters, and 0.75 mm. for each of the following 4 diopters.

EFFECT OF IGNIPUNCTURE

Clinical Observations.—Knowledge is meager regarding the changes in the eye after ignipuncture, because (*a*) immediately after the operation the eyes are bandaged and are not examined for about a week, (*b*) a microscopic study of enucleated human eyes following ignipuncture has not been made and (*c*) very little experimental work has been done along this line. Since it has been my invariable rule to make three ophthalmoscopic examinations—one immediately before the sclerochoroidal incision, one directly after it, and a third after the ignipuncture—I have as precise an idea of what happens as the ophthalmoscope can reveal.

The preliminary scleral puncture, followed by a more or less copious flow of vitreous, usually brings about an apposition of the retina to the choroid, provided that the subretinal fluid is not yet organized or albuminous, as it is in detachments of long duration. The introduction of the red hot cautery has a number of results: It cauterizes the sclera, choroid, retina and adjoining vitreous. It probably has some effect on the nervous system of the uvea. It diminishes the volume of the eyeball. The ophthalmoscopic picture is characteristic. One sees a dark round depression, the hole in the three structures of the wall of the eyeball. Around this hole there is a grayish-white zone, about 2 or 3 diopters in diameter, corresponding to the cauterized retina. The vitreous in front of the hole is turbid. At times the retina shows folds extending in various directions from the area of ignipuncture. In one case a number of whitish spots appeared in the retina opposite to the fundus in which ignipuncture had been performed. This was attributed at the time to the effect of irradiated heat from the cautery tip. The picture usually remains unchanged for a few weeks; then the dark hole begins to become occluded by a grayish layer, the edge of the retinal hole flattens out and considerable pigment accumulates at this edge and also over a wide area surrounding it. In the course of weeks or months, in the

place of the hole there is a white patch surrounded by irregular depigmented areas alternating with other areas strongly pigmented. This description applies to the cases in which ignipuncture succeeded in anchoring the retina to its proper place.

EXPERIMENTAL DATA

A study of rabbits' eyes enucleated at various intervals after an ignipuncture furnishes interesting information.[2]

Eye 1.—This eye was enucleated five minutes after an ignipuncture. Looking at the punctured area from the retinal surface one saw a hole about 1.5 mm. wide. Underneath the clearcut edge of the retinal hole the edge of the hole in the choroid was seen, and behind this the edge of the scleral hole. The vitreous, which had prolapsed into the cavity of the hole, was turbid and striated and appeared like a broad strand extending across the hole, with one of its extremities adherent to one edge while the opposite extremity was lost in the surrounding vitreous. In front of the hole there was a grayish mass, an area of cauterized vitreous.

Eye 2.—This was enucleated five days after ignipuncture. The choroid and retina became detached from the sclera in this specimen (artefact?), and this accident offered the possibility of obtaining a good view of the outer surface of the choroid and retina as well as of their inner surface. The hole, which was about 1.5 mm. in diameter, was visible on the outer surface of the area of the choroid in which ignipuncture had been performed; its dark-pigmented edge was surrounded by a pale ring, this by a pigmented zone and the latter by a pale zone; that is, the choroid reacted to the ignipuncture by a shifting of pigment into two concentric dark rings. The center of the hole in the choroid was occupied by a whitish-gray, soft looking mass. Folding back the choroid, so as to look at its inner surface, one saw the hole surrounded by an intensely white zone, 2 mm. wide. One also obtained a good view of the outer surface of the retina which showed the clearcut edge surrounding the hole. Part of the hole in the retina was plugged by a mass of vitreous. Some of this mass was adherent to the retina. The inner surface of the retina showed the hole with its sharp gray edge (coagulated retina) and the turbid mass of vitreous prolapsed into it. In the retina the edge of the hole was somewhat larger than that in the choroid, and the exposed choroid was covered by chalk white broken-down tissue. The retinal ring was about 1.5 mm. in diameter. The vitreous plug extended throughout the center of the hole and adhered on one side to the choroid and the edge of the retinal hole. The vitreous adjoining the retinal hole was disturbed, gray and opaque, and it contained strands and pseudomembranes.

Eye 3.—This eye was enucleated twenty-one days after the ignipuncture. The inner surface of the area in which ignipuncture had been performed showed an irregular quadrangular depression, 2 by 3 mm. in size, surrounded by a more or less concentric, narrow, whitish ring, 4 or 5 mm. in diameter. The zone between the margin of the former hole and the pale ring was slightly depigmented. The outer edge of the white ring was dark. A white strip, 1.5 mm. wide, extended from the hole in the retina to the optic disk. The slit-lamp clearly showed how

2. The area involved is excised from the eye, immersed in water and examined with the slit-lamp and microscope. (A full description of this macromicroscopic method will appear in a special paper.)

the hole had been occluded by a thin whitish layer. Adherent to this was a conical conglomerate of vitreous strands projecting its narrow point into the vitreous. The lens showed a white discoid patch on its posterior surface, near the equator. The capsule showed a slit through which some cortex protruded. This was the result of the tip of the cautery having touched the lens.

Comment.—Although no analogy exists between a rabbit's healthy eye and a human eye with a retinal detachment, one may compare with profit the changes that occur in these eyes after ignipuncture. The hot cautery tip burrows a hole through the sclera, choroid and retina and enters the vitreous, producing definite and almost similar reactions. Surrounding the hole in the choroid there is a white ring around which the pigment has more or less disappeared. This pigment wanders in a centrifugal direction and accumulates in patches or in a ring around the pale zone. The retina shows a similar whitish-gray zone surrounding the hole. The vitreous is coagulated in the vicinity of the hole, and part of it prolapses into the opening made by the cautery; gradually the hole closes, and the vitreous becomes adherent to the cauterized area. All this corresponds fairly well with what is seen with the ophthalmoscope in human eyes. The importance of these observations lies in the light that they throw on the entire idea behind the Gonin operation. They enable one to understand better what one is aiming at. If the treatment is to succeed, one has to achieve the following results: (1) produce an inflammatory reaction in the choroid; (2) anchor the retina to the underlying choroid; (3) cauterize the vitreous so as to obtain a plug that will obstruct the hole in the retina, prolapse into the opening and become adherent, at least in part, to the edge of the hole and later to the tissue that grows over the opening.

COMPLICATIONS

The complications so far encountered during and after the Gonin operation are: (1) hemorrhages in the vitreous, (2) collapse of the eyeball, (3) fistula of the area in which ignipuncture is performed and hypotension, (4) iritis and (5) relapses.

The entrance of blood into the vitreous may be (*a*) from the episcleral vessels if the surface of the exposed sclera is not constantly kept dry during the operation; (*b*) from the long ciliary arteries; (*c*) from the choroid; in the latter case the hemorrhage may occur during the sclerochoroidal puncture or during convalescence; (*d*) from the ciliary region and (*e*) from the retinal blood vessels.

The blood originating from the choroid and ciliary region appears as dark streaks or sheets of infiltration between the various strata of vitreous, as a dark curtain hanging immediately behind the lens obstructing entirely the entrance of light into the fundus and excluding com-

pletely the presence of a red reflex or as a dark red mass occupying the lowest levels of the vitreous. Most of the hemorrhages into the vitreous occur during the first two weeks following the operation. Complete rest is perhaps the best preventive measure.

The hemorrhage originating from a retinal blood vessel usually has the appearance of round red spots or long, sausage-shaped red blotches.

Collapse of the eyeball following scleral puncture occurs when the loss of vitreous is relatively large and if the tension of the eyeball is low before the operation. This complication is unpleasant in that it makes the introduction of the cautery tip difficult. In the course of a few weeks the eyeball usually recovers its normal tension. When the scleral hole is open for some time, the eyeball remains soft, and vitreous fluid infiltrates the subconjunctival space. In one case this complication lasted for about ten days. It disappeared without leaving any untoward result.

In one case a patient complained of severe pain in the eye on which the operation was done; it appeared about the third day after the operation and lasted for about two weeks. He volunteered the information that he had a similar attack of iritis after the first ignipuncture, which was done the previous year.

A relapse had occurred in three cases. The first patient was successfully operated on by Professor Gonin, about a year before the present writing, for a retinal detachment in the upper temporal quadrant of the fundus. About nine months later there appeared a detachment with a tear in the lower part of the fundus. The second patient, operated on successfully by Dr. S. Slomka of New York, recovered a vision of 20/30 (he had had perception of hand movements before the operation) and remained well for about seven months. Then a new tear appeared, and the retina began to become detached over a small area adjoining that of the previous ignipuncture.

REPORT OF CASES

Relapse.—Case 1.—Edward G., aged 67, was referred by Dr. H. S. Miles of Bridgeport, Conn. In 1922, a retinal detachment in the left eye resulted in complete loss of vision. In May, 1929, a similar condition developed in the right eye, with a tear in the upper temporal sector of the fundus. Ignipuncture was performed by Professor Gonin in October. The vision, which had diminished to perception of hand movements, gradually improved to about 8/200 after the operation. This condition remained stationary until August, 1930, when a new retinal detachment developed in the lower part of the fundus, reducing vision to perception of light. An ignipuncture improved the condition to 10/200 or perhaps better, but the retina did not become entirely reattached.

CASE 2.—Mrs. S., aged 45, had a detachment of the retina and a hole in the macula of several months' standing. The retina became completely reattached after trephining of the sclera over the area corresponding to the highest bulge of the retina. About one year after the operation, the retina became detached again. Two ignipunctures were unsuccessful.

CASE 3.—Mr. V. L., a patient of Dr. S. Slomka of New York, aged 57, was first seen on April 10, 1930. Five years before, the left eye became blind from a retinal detachment caused by an injury. Ten days before examination the patient fell off a scaffold (as on the former occasion); a day or two later he noticed that the vision of the right eye was blurred. Examination revealed some fine vitreous opacities. There was a shallow detachment of the upper temporal quadrant of the retina with a tear, about 1 disk diameter in size and about 2 disk diameters from the ora serrata at about 11 o'clock. Vision was 20/40—. The visual field had a corresponding contraction in the lower nasal periphery.

In the left eye there was an old, practically total detachment of the retina. Vision was limited to perception of hand movements in the lower field. By May 3, 1930, the detachment in the right eye involved the entire temporal part of the retina; vision was 1/200. On May 9, ignipuncture was performed by Dr. Slomka, with the result that the retina was reattached; vision was then 20/70. On June 9, on rising the patient noticed his vision to be somewhat blurred. Examination disclosed a shallow detachment of the retina in the upper periphery, extending also temporally, and some fresh punctate retinal hemorrhages. In the macula there were several horizontal shallow retinal folds. In the extreme temporal periphery, at about 9 o'clock, the retina appeared honeycombed. The old tear was closed by scar tissue. Vision was 20/200. The visual field was contracted nasally and below. On June 11, another Gonin operation was performed, with the result that the retina became reattached and the folds in the macula disappeared. Vision was 20/50 and in a short time improved to 20/30—, without the aid of glasses. The visual field was somewhat contracted in the lower nasal periphery. The patient was examined at frequent intervals and the condition remained stationary. On March 3, 1931, a U-shaped tear surrounded by small retinal hemorrhages was discovered immediately posterior to the first cauterized area. There was a small area of retinal detachment. Vision was still 20/30. At this date (August, 1931) the condition is still the same.

Improvement.—Patients are to be considered improved when the acuity of vision and the size of the field show a decided increase, though the retina does not become entirely attached. Several such cases were observed.

CASE 4.—Mr. A., at the age of 49, lost the vision of his left eye due to a retinal detachment. Eleven years later, a similar condition developed in the right eye, and vision was reduced to perception of hand movements in the upper part of the field. Four Gonin operations were performed two years after the beginning of the trouble. His field of vision improved to the extent that he could walk alone, and his visual acuity improved to ability to read the first three lines of the Snellen chart at about 2 to 3 feet (61 to 91 cm.). He had maintained his visual gain when he was last examined (about one and a half years following the last operation).

CASE 5.—Mrs. P., aged 24, was operated on by Dr. A. Knapp for a retinal detachment in the left eye, in which vision was reduced to 3/200—. The ignipunc-

ture anchored the retina on the temporal side, and vision improved to 20/70—. Fourteen months later the lower nasal part of the fundus still showed a shallow detachment, but the improvement in vision and field remained unchanged.

CASE 6.—Paul K., aged 29, who was referred by Dr. I. J. Kruskal, of Brooklyn, had myopia of about 25 diopters. At the age of 26 he had lost vision in the right eye on account of a traumatic detachment of the retina. Two days before examination, a retinal detachment appeared in the left eye. Vision was reduced to counting fingers at 10 cm. in the upper part of the field. The retinal detachment occupied the entire upper and temporal part of the fundus. Several tears, some larger than from 1 to 2 disk diameters, were seen above and below temporally, very far behind the equator. The retina showed several folds and areas of cystic degeneration, and the vitreous was full of dusty and flocculent opacities. The eyeball was very large. Several opacities of the lens, with the turbidity of the vitreous, made the study of the fundus difficult. After three ignipunctures the vitreous began to clear up, and the upper and temporal portions of the fundus regained a normal appearance. The retina remained reattached, except in the lower part. Seven months after the second ignipuncture the patient's vision attained 6/200.

The remarkable feature about this case is the relatively great improvement obtained in a condition that looked hopeless, an improvement that began to appear a number of months after the second ignipuncture. This late improvement occurred in a few other cases under my observation. The second ignipuncture had to be made 27 mm. behind the limbus. A preliminary canthotomy was necessary to make the area accessible.

Failures.—To the cases of failures reported in my previous paper there are many more to be added. Most of them belong to the group of detachments in which no tears were discovered or in which the tears were too large. Operation was performed in order to find a way of handling such cases. Some of the cases are worth reporting on account of some unusual features.

CASE 7.—J. T., a girl, aged 12, a patient of Dr. John Wheeler of New York, began to wear glasses (—4 sphere) at the age of about 2½. During the following eight years her myopia increased so that at the age of 9 she needed a —11 sphere. When she was 10 years old a total retinal detachment and cataract developed in her left eye. Two years later the retina became detached in the right eye; at examination one could see a dialysis of about one fourth of the circumference of the retina, occupying the ora serrata in the upper temporal quadrant. Within the next few days the dialysis extended still farther, and the free edge of the retina looked curled on itself. The vitreous showed marked signs of degeneration and the eye was soft. After one ignipuncture the retina seemed to arrange itself so that its free edge could scarcely be seen. The eye did not react well, however, to the ignipuncture. A clouding of the deeper layers of the vitreous and collapse of the ocular tension followed the surgical intervention. After several weeks there was slight improvement, but it was apparent that an eye with this condition is not suitable for a Gonin operation.

CASE 8.—In Mrs. M., a patient of Dr. H. Tyson of New York, following scleral puncture and the escape of subretinal fluid, the ophthalmoscope revealed a

number of patches of active choroiditis. The part of the sclera corresponding to the pathologic choroidal area appeared dark and slightly bulging. Although the acuity and field of vision improved considerably (from perception of light with poor projection to counting fingers at about 10 feet [304 cm.] after operation), the improvement lasted only a short time. The retinal detachment became more extensive. A second ignipuncture had no effect.

Another case that appears to be a failure is interesting on account of the special problem that it offered:

CASE 9.—Mr. H. R., aged 33, a patient of Dr. Terry Smith and Dr. A. L. Prince of Hartford, Conn., presented a retinal detachment occupying the temporal half and lower part of the fundus of the left eye. In the upper part there were several spots which looked like retinal holes that had become obstructed. In the lower part of the fundus there were two distinct holes in the bulging retina. The decision was taken to apply the ignipuncture to the lower part of the fundus. Following this treatment the retina flattened out and became partly reattached in the lower part (the cloudy vitreous did not allow a good view), but at the same time the retina became detached in the upper part of the fundus and a small dialysis appeared above and temporally in the region of the ora serrata. The question is whether it would not have been wiser to apply the first ignipuncture in the upper part of the fundus.

Good Results.—The following case presents an unusual feature in that it twice showed a detachment of the retina which reattached itself spontaneously and finally appeared in the same area the third time. It became so extensive that a Gonin operation had to be performed.

CASE 10.—Bernard K., aged 19, a college student, complained on March 29, 1930, of a blur in the lower nasal field of the left eye. Vision was 20/20 in the right eye with a —4 sphere —50 cylinder axis 90, and 20/20 in the left eye with a —4 sphere —50 cylinder axis 180. Examination revealed a detachment of the retina occupying about one fourth of the upper part of the fundus with a slitlike tear situated very near the ora serrata in the direction of 12 o'clock. The vitreous showed characteristic dustlike opacities and the retina involved exhibited a number of pale, small, faint striae and foci. After a rest in bed for a few days, he returned on April 3, with complete disappearance of the detachment. The tear could be seen as a faint pink spot. On April 18, he returned with a somewhat more extensive detachment in the same location. After a stay of six days in bed the retina became reattached. This condition remained unchanged until June 18, when he returned with the retina detached in the same area. This time the detachment extended temporally downward to a level of about 2 disk diameters above the macula. A few cystic formations in the retina appeared midway between the equator and the macula. He was admitted to the Knapp Memorial Hospital and an ignipuncture was performed on July 15. The result left nothing to be desired. Reattachment was complete and vision normal when he was last examined (August, 1931).

CASE 11.—In William S., a merchant, 47 years of age, with a myopia of 7 diopters in the right eye and 6 diopters in the left, a spontaneous retinal detachment developed in the lower temporal sector of the fundus of the right eye on Jan. 2, 1930. There were chorioretinal patches at the periphery, a small atrophic patch in the macular area, a fluid vitreous and a few dustlike opacities. During

four weeks of rest in bed the detachment extended over a larger area, and in addition a small tear appeared in the retina on the temporal side at 9 o'clock. Vision diminished from 20/50 to 5/200. An ignipuncture performed in February, 1930, resulted in a reattachment of the retina and restoration of the original vision of 20/50+. This patient has since been examined repeatedly. At the last examination, in August, 1931, the condition was still satisfactory.

It is interesting to note in case 11 that the tear in the retina appeared about three weeks after the appearance of the detachment; consequently, it could not be considered as the cause of the detachment. On the other hand, the ignipuncture was aimed at this tear. It struck it, and the detachment disappeared. Would cauterization of the choroid and retina alone, without even striking the edge of the hole, have been sufficient to arrest the process?

CASE 12.—In Mrs. S. D., aged 54, a patient of Dr. LeGrand H. Hardy of New York, a retinal detachment developed on Jan. 5, 1931. It involved the upper half of the fundus and there was a small tear near the ora serrata. This condition followed a so-called "bilious attack," during which the patient vomited about ten times. Ignipuncture was performed within one week after the appearance of the retinal detachment, and the result was most satisfactory. Before operation vision was 20/100 in the right eye and 20/20+ in the left. At present it is 20/20— in the right eye and 20/20+ in the left. The visual field is good.

CONCLUSIONS

1. The Gonin method of treatment for retinal detachments is and will remain on probation for a considerable length of time.

2. In the selected cases this method gives surprisingly good results.

3. The two main difficulties to be mastered thoroughly by the surgeon undertaking to apply this method are (1) the selection of the proper cases, (2) carrying out the proper technic.

4. Early operation increases the chance of recovery.

5. Securing beforehand the patient's cooperation and his determination to go through the lengthy treatment is essential.

A CHECKING STANDARD FOR TONOMETERS*

C. E. FERREE, PH.D.
AND
G. RAND, PH.D.
BALTIMORE

There seems to be no type of test or measurement in the practice of ophthalmology more acutely in need of standardization than tonometry. Among the essentials for standardization the following may be mentioned: 1. A clearer conception of what is measured in tonometry. 2. Preferably the adoption of one type of instrument as the standard of practice. 3. A determination of all the variable factors that influence the measurements in tonometry. 4. Devices and methods for the control of these factors or for reducing their influence to a minimum. 5. The better care of tonometers. 6. The devising of a checking standard by means of which the performance or reading of an instrument at any one time can be compared with the performance at any other time or with that of any other instrument of its type. 7. The careful selection of an instrument of a given type and the determination with it of distribution curves for a sufficiently large number of eyes, normal, abnormal and suspicious. This instrument should be carefully preserved as a standard, and its readings compared with the readings of other instruments by means of the checking standard for the recalibration of the scales of those instruments or for the determination of correction factors.

Instruments of the Schiötz type are designed to give an indirect measure of the resistance of the cornea to external pressure. Intra-ocular pressure is an important, but not the only, factor in this resistance. The instrument is so constructed that the depth of the indentation of the cornea under different pressures is registered on a scale that sustains a direct relation to the resistance of the cornea. With a proper control and specification of conditions, it should be sufficient and much more simple to express the pressure applied in terms of dynes per square centimeter or square millimeter, or in units of the centimeter-gram-second system, and to measure the indentation directly in terms of millimeters. A diagnostic scale or distribution curve could then be formed directly in terms of depth of indentation instead of a somewhat

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* From the Research Laboratory of Physiological Optics, Wilmer Ophthalmological Institute, Johns Hopkins Medical School.

hypothetic intra-ocular pressure. One of the most confusing features of Schiötz' plan of measurement is his attempt to translate the scale readings of the instrument into intra-ocular pressures.

In the construction of a checking standard, however, it has not been our purpose to make the digression suggested. There has been no attempt to reduce the measurement to absolute terms. The device is limited to a comparison under standard conditions of the readings of the same instrument at different times, or of one instrument with those of another or with those of one instrument preserved as a standard. While a claim to absolute measurement has been made for checking devices of the manometer type for various purposes other than the one described here, such claims are not deemed by us entirely sound for any device in which a rubber or thin elastic membrane is required to play an invariable rôle through a period of time. It is safer to use such devices for comparative measurements only.

In case of the present device,[1] the pressures needed are obtained by means of a column of mercury of variable height. A water column, such as was used by Schiötz, for example, because of the lower specific gravity of water, would have to undergo much greater changes in height to produce given changes in pressure than a column of mercury, and would thus offer the possibility of greater sensitivity in calibration. To give the same range of pressure as a column of mercury 150 mm. high, however, a column of water would have to be raised through a range of nearly 2,000 mm. or 2 meters. This would make the instrument unduly cumbersome and unwieldy to operate and to handle. In the present instrument even greater sensitivity of calibration than is ordinarily had with a column of water is allowed by making the height of the column of mercury micrometrically variable and measurable, and at the same time the instrument is given compactness, portability and great ease and convenience of operation.

In general outline the instrument consists of the following members or parts: 1. A glass tube (pyrex buret tubing), 4 cm. long, carefully selected with reference to uniformity of bore, at the top of which is a structure of dimensions, parts and shape to stimulate the outer coat of the eye, sclerotic and cornea. 2. A second glass tube, 18 cm. long, also selected for uniformity of bore throughout. In this tube the height of the column of mercury is varied to give the amounts of pressure that are needed against the artificial cornea noted in 1. The tube is graduated in millimeters to serve as a check on the micrometer scale. 3. A block of cold rolled iron, 5 by 5 by 10 cm., in which is a cylindric hollow core, 2 cm. in diameter and 10 cm. long. At a point near each end of the block are

1. This instrument was carefully and accurately constructed by our instrument maker, A. H. Goebel.

vertical cylinders of steel of suitable height into which are fitted tightly the tubes noted in 1 and 2. 4. A piston for applying pressure to the mercury in 3, to vary the height of the column in 2 and to vary by corresponding amounts the pressure of the mercury against the membrane simulating the cornea in the eye-shaped bulb at the top of 1. 5. A micrometer screw to displace the piston noted in 4, and a scale calibrated to read the change of height of the column of mercury noted in 1 to fifths of a millimeter. 6. A second piston for changing by any desired amount the relation of the height of the column of mercury to the reading on the micrometer scale of the piston in 4. If desired, this piston can be used also to make smaller changes in the height of the column of mercury than can be made by the piston in 4. 7. A micrometer screw to displace this piston and a scale calibrated to read a change of height of the column of mercury in 1 to fiftieths of a milli-

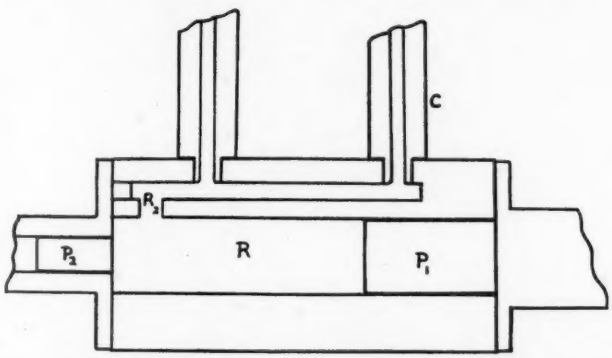

Fig. 1.—Checking standard for tonometers, longitudinal section.

meter. 8. A supporting structure to hold the tonometer in a predetermined fixed relation to the artificial cornea. 9. A tripod base on which the various structures noted are mounted.

A longitudinal section of the instrument is shown in figure 1, and a photograph of the front and rear views in figures 2 and 3, respectively. At T_1 in figures 2 and 3 is the glass tube carrying at its upper end the eye structure. The diameter of this tube is 10 mm. The horizontal diameter of the artificial eye is 23 mm.; the depth along the vertical diameter is 21.3 mm. At the top is a circular opening, 12 mm. in diameter, to approximate the diameter of the base of the cornea. Around the edge of the opening, the wall of the structure is sharply flared to give a rim about 1 mm. broad as an aid in the secure attachment of the membrane simulating the cornea. This membrane is made of rubber sheeting (thin dental dam), shaped to give under upward pressure a convexed surface as closely resembling that of the cornea as possible. For pressures up to 91 mm. of mercury (Schiötz,[2] 1925 scale)

2. Schiötz, H.: Tonometry, Brit. J. Ophth. **9**:145, 1924.

a membrane 0.0146 mm. thick is used. For pressures greater than this it has been found necessary to use a heavier membrane, e. g., 0.0292 mm. thick. Under the greater pressures the thinner membrane was found to distend too much and to undergo too great a change of shape. Also for these pressures the heavier or thicker membrane gives a higher reproducibility of result. Before this membrane is fastened in place, mercury is poured into the system consisting of the two glass

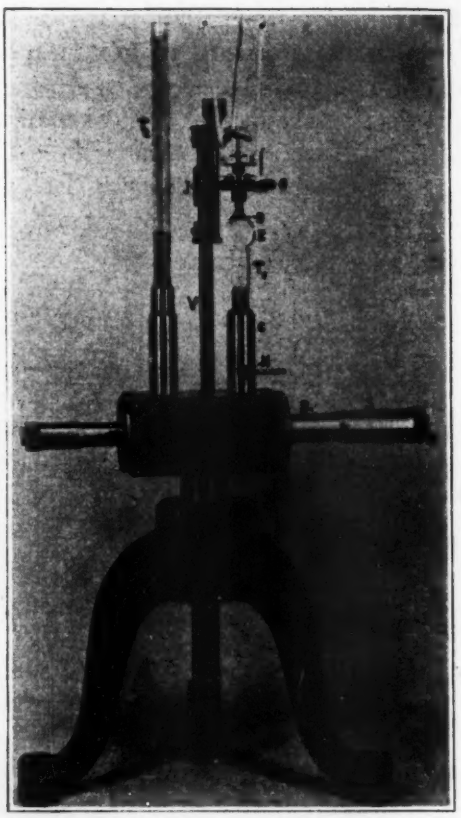

Fig. 2.—Checking standard for tonometers, front view.

tubes, the supporting steel cylinders and the cylindric reservoir, until it rises to the height of approximately 2 cm. in each of the glass tubes. Tube T_1 is then filled with water to the line of the brim of the eye structure, and the membrane, which is cut large by the correct amount, is fastened across the top, leaving a sufficient amount of slack to allow it to become convex under pressure without too much stretching. Water is then poured on the top of the membrane to insure that its lower surface is pressed into contact with the water beneath. Pressure is then applied to the mercury in the cylinder by means of the micrometer screw

until the amount is secured that is chosen as the starting point of the calibration. In doing this, great care is exercised that no air bubbles remain between the surface of the water and the membrane. At *E* is shown the eye bulb, and at *D* the diaphragm or artificial cornea. At P_1 is shown the piston in its casing noted in 4, and at *H* the circular head, the turning of which drives the micrometer screw. As the screw moves forward, it causes the sleeve *S* to telescope over the barrel *B*. On the

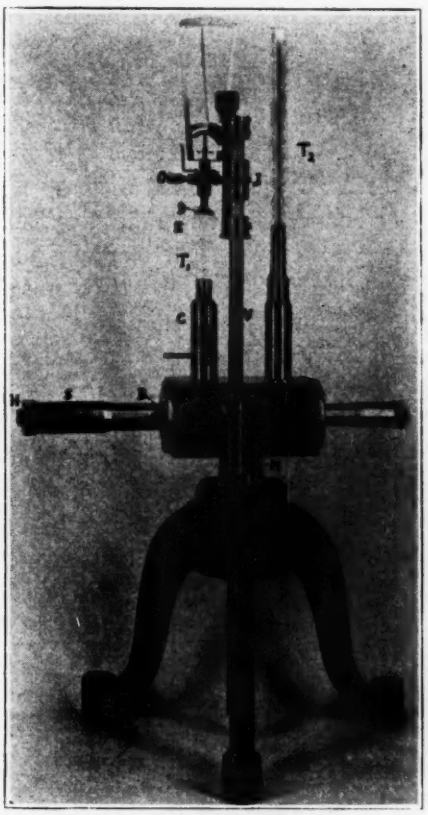

Fig. 3.—Checking standard for tonometers, rear view.

barrel *B* is a linear scale graduated in fourths of a centimeter. On the beveled edge of the sleeve *S* is a circular scale graduated in fifty equal divisions. The diameter of the piston is 2 cm.; its length, 3.5 cm.

At T_2 is shown the second glass tube. This tube contains the column of mercury the height of which is varied to give the different amounts of pressure needed against the diaphragm *D*. The length of this tube is 180 mm.; its diameter, 10 mm. Great care was exercised in the selection of tubes T_1 and T_2 in order to secure uniformity of bore throughout. A great number of tubes had to be examined before two could be found

suitable for use. To aid in the examination of these tubes, a gage was made consisting of a metal disk, exactly 10 mm. in diameter, mounted at the end of a rod of suitable length. The test was made by passing the disk through the tube from one end to the other. In the planning and graduation of tube T_2, provision was made that the height of the column might be made to vary through 160 mm. This variation has to be accomplished by the displacement of mercury by the piston P_1. On the ratio of the area of the cross-section of tube T_2 and that of the piston P_1, then, will depend the distance the piston has to move. This ratio was chosen as 1 : 4; i. e., the diameters of the tube and piston were made in the ratio 1 : 2. With these ratios, then, in order to make the column of mercury in the tube vary through 160 mm., the piston would have to move through 40 mm. The ratio 1 : 4 is also an important factor in determining the pitch of the screw that drives the piston through the cylinder. That is, for convenience in graduating, an integral number of turns should be required to cause the mercury in tube T_2 to rise an integral number of units on the scale. By choosing a pitch of four turns in 1 cm. or one turn in 2.5 mm., one turn of the screw causes the column of mercury in tube T_2 to rise 10 mm. By graduating the circular scale on the beveled edge of the sleeve S in fifty divisions, then, one division corresponds to a change of one fifth of a millimeter in the height of the column of mercury in tube T_2, and by graduating the scale in one hundred divisions, readings can be made to one tenth of a millimeter without interpolation. Fineness of reading could also be attained by a smaller number of divisions on the sleeve S and the use of a vernier attachment. The present piston is graduated to read to fifths of a millimeter without interpolation. As a check on the readings on the micrometer scale, the tube T_2 is graduated in millimeters. On this scale the height of the column of mercury can be read directly in millimeters and, by interpolation, in quarter millimeters. In close contiguity with the millimeter scale there is also a volume scale graduated in milliliters. The purpose of this scale is, by comparison with the millimeter scale, to serve as a check on the uniformity of bore in the various parts of the tube.

The cylindric reservoir, which holds the residual supply of mercury and in which the piston P_1 moves, is shown at R in figure 1. It is 2 cm. in diameter and 10 cm. long. The total volume of mercury used is 32 cc. This reservoir communicates with the tubes T_1 and T_2 by a small cylindric right-angled passage R_2, as is shown in figure 1. The cylinder C, which supports the tube T_1, is fitted with a stop-cock, shown at N in figure 2. When the passage in C is closed by means of this stop-cock, the pressure in tube T_1 can be held constant at any desired value. The stop-cock is held from slipping in and out, when turned, by a steel spring

or lamina, one end of which is screwed to the wall of the cylinder. The other end, which is V-shaped, fits between a button and a shoulder on the projecting end of the stop-cock.

The second piston, which is used to change the relation between the readings on the scale on tube T_2 and the micrometer scale for piston P_1, or for making finer changes in the height of mercury in tube T_2 than can be made with piston P_1, is shown at P_2 in figure 1. This piston fits into a cylinder, 1 cm. in diameter and 2 cm. long, which is mounted in alinement with reservoir R at the end opposite to piston P_1. At its zero position this piston lies entirely outside of reservoir R. Its forward thrust carries it into reservoir R to a depth of 2 cm. It is provided with a short micrometer screw with a pitch of 1 mm.; it is also provided with a barrel with a linear scale graduated in millimeters and a sleeve, the beveled forward edge of which is graduated in fifty equal divisions. The diameter of this position is 1 cm.; its length, 2 cm. Since the diameter of this position is the same as that of tube T_2 and the pitch of the driving screw is 1 mm., one turn of the screw causes the mercury to rise 1 mm. in tube T_2; a turn through one division of the circular scale on the forward edge of the sleeve, one fiftieth of a millimeter. By means of this piston the point that is to serve as the zero on the scale on tube T_2 can, for example, be shifted at will without disturbing the reading on the scale for piston P_1. This change can be used for purposes of correction when for some reason the zero has been shifted, or it can be used to establish a new zero. It could also be used, for example, to make the readings on the scale of piston P_1 correspond at some point to a given value in Schiötz' scale selected as a starting point in a calibration in terms of millimeters of intra-ocular pressure. And, as already stated, it can further be used to make smaller changes in the height of the mercury column in tube T_2 than can be made by piston P_1.

The instrument is mounted on a tripod base. To give stability of support, this base is of wide spread and is extra heavy. To allow for adjustment of height the center of the base is raised, cored out to receive the heavy vertical rod or stem of the instrument and provided with a set screw. On the vertical bar just below the iron block that contains the reservoir of mercury is mounted the support for the tonometer under examination. This support consists of the following parts: a horizontal member M, a vertical member V, a carrier J, a clamp or holder O for the tonometer and provisions for a three-dimensional adjustment. The horizontal member M consists of an outwardly extending arm fitted at its inner end with a collar and set screw, which serve as an attachment to the stem of the instrument, and at its outer end with a socket and set screw, which serve as a support for the vertical member. The vertical member V comprises an upwardly extending rod

and a construction for the attachment and adjustment of the carrier *J*. This carrier is a broad collar which travels up and down the vertical rod. Attached to this carrier by means of a heavy band is a flat piece of metal, the farther end of which is shaped to form one of the jaws of the holder *O* for the tonometer. Through this piece of metal near its base is threaded a large screw which is fixedly attached at top and bottom to the vertical rod by heavy bands or collars. This screw is provided with a milled head. It raises and lowers the carriage and provides the adjustment for height. The holder or clamp *O* consists of two sloping jaws, one movable and the other fixed. The movable jaw is fastened to the fixed jaw near its base by means of a hinge. The jaws are opened and closed by a stirrup-shaped device and a set screw. The set screw is threaded through the foot piece of the stirrup, and the stirrup itself straddles the movable jaw and is fastened by a swivel to the fixed jaw at a suitable distance from the bite or grip, which holds the tonometer. Two adjustments are thus made possible: The set screw can be tightened or loosened, and it can be moved by means of the stirrup along the sloping edge of the jaw as along a cam. Either or both of these adjustments serve to open or close the jaws of the holder. The adjustment of the position of the plunger on the diaphragm *D* is accomplished as follows: The horizontal arm can be rotated about the stem or polar axis of the instrument and clamped in any desired position; also the vertical rod can be turned about its axis and clamped where desired. Each of these motions causes the tonometer to describe an arc, and the construction is such that by the proper combination of the two motions the arcs described by the center of the foot of the plunger can be made to intersect in the vertical axis of the tube T_1 or in the vertical line passing through the center of the diaphragm *D*. The correct placement of the plunger on the diaphragm *D* is completed by making the required adjustment for height.

CONCLUSIONS

In conclusion, it is hardly needful to point out that a checking standard is as badly needed for tonometers as it is for photometers, radiometers, meter rods, foot rules or any other instruments or devices used for measurement. No one would think of using a photometer provided with a calibrated scale, for example, without frequent reference to a checking standard. Even instruments of the same type made to specification are apt to vary, particularly if the manufacturers are not provided with adequate means for testing and checking their performance. Also, delicate instruments in frequent use are almost sure to change unless very great care is taken of them. In a survey of the literature it is amazing to see how much work and

ingenuity have been expended on the devising of tonometers of different kinds and how little attention has been given to means for checking them for constancy and uniformity of performance. As Schiötz has aptly remarked, the human eye cannot be used as a checking standard for tonometers. If this paper accomplishes no other results than to call attention to the fundamental need for checking standards and the possibility of obtaining them, we will be sufficiently repaid for our efforts.

Another possible use of the instrument and, it would seem to us, a valuable one, is as a model on which beginners could acquire practice in the correct placement, holding and reading of the instrument, and in securing reproducibility of result. To have to acquire on the human eye all the manual skill and deftness that is required in the successful use of the tonometer is unfortunate both for the patient and for the examiner. Furthermore, the only check that an examiner has on the correctness of his technic is reproducibility of result. In case of the eye, this check is greatly interfered with because of the variability in the resistance that is offered by the cornea and other structures and parts of the eye and by the contents of the eye during a series of measurements. It would seem clear that a certain amount of practice should be had under conditions that can be held constant, and certain features of the technic should become familiar and more or less automatic before the more difficult work on the eye is begun. This can be done quite readily and effectively, we believe, with the instrument described in this paper.

INTRASCLERAL NERVE LOOPS*

ALGERNON B. REESE, M.D.
NEW YORK

The long ciliary nerves accompany the long posterior ciliary arteries through their emissaries in the posterior part of the sclera, each nerve dividing into two branches before emerging into the suprachoroidal space. These four nerves course forward in a meridional direction between the sclera and the choroid. Sometimes near the junction of the orbiculus ciliaris and the corona ciliaris, or from 2.5 to 3 mm. from the angle of the anterior chamber, the ciliary nerve enters the sclera and then turns abruptly back again to resume its forward course into the ciliary body. Occasionally the nerve loop thus formed extends through the entire thickness of the sclera with its cupola projecting above the external scleral surface, covered only by conjunctiva. This nerve loop may extend into the sclera perpendicular to, or at an angle with, the surface, or it may be retroverted or anteflexed. Its apex is sometimes mushroom-shaped, due to a neuroma-like enlargement. Chromatophores from the suprachoroid appear at times extending up into the interspace of the loop, and occasionally similar pigment cells as well as smooth muscle from the ciliary body are seen along the wall of the scleral foramen through which the loop extends. On rare occasions a cyst is seen accompanying a loop. The anterior ciliary artery in its inward course may share the emissary with the nerve loop, or the blood vessel accompanying the loop may be an anastomotic branch between the episcleral and the ciliary vessels. Either the cupola or the stem of the loop may send off nerve branches which extend forward into the cornea.

In six of the seven cases that I have examined clinically, the loop has been in the horizontal plane on the nasal side of the globe and in one case in the vertical plane inferiorly. One patient showed a loop in each eye nasally. However, these loops may appear temporally or superiorly, and Fritz [1] claimed that a common site is superiorly under the insertion of the superior rectus muscle. Reports on their frequency in these various sites have been for the most part based on microscopic specimens and are misleading, as practically all globes are sectioned in

* Submitted for publication, Aug. 20, 1931.

* Read at a meeting of the American Ophthalmological Society, Asheville, N. C., June 1, 1931.

1. Fritz: Sitzungsb. d. k. Akad. d. Wissenschaft Math.-naturw. cl., Wien. **113**:273, 1904.

Fig. 1.—An intrascleral nerve loop as it appears clinically.

the horizontal plane. E. Fuchs [2] reported on thirteen eyes examined microscopically in which the finding of the loops was incidental. In eleven of these the loop extended entirely through the sclera and projected above the external scleral surface; it therefore could unquestionably have been seen clinically. Nine showed a neuroma-like enlargement of the cupola; five were accompanied by anterior ciliary arteries; three showed loops in the posterior part of the sclera near the nerve's entrance through the emissary, and one eye showed two loops. Fischer [3] reported on forty loops seen in microscopic preparations, of which thirteen showed complete perforation of the sclera to a point beyond the external scleral surface and twenty-seven only partial perforation. Seven loops were accompanied by the anterior ciliary artery, and one was in the posterior part of the sclera near the emissary

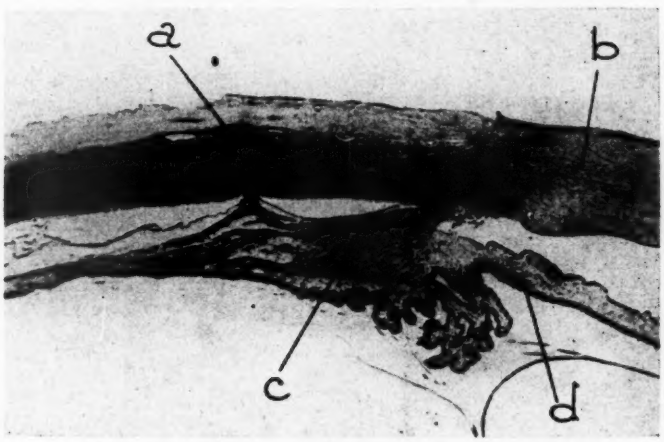

Fig. 2.—A nerve loop extending through the sclera just to the external surface: *a*, nerve loop; *b*, cornea; *c*, ciliary body, and *d*, iris.

of the nerve. One eye had three loops. In a 14 year old patient Kyrieleis [4] reported the growth, superiorly at the site of a loop, of a tumor which proved on microscopic examination to be a neuroma and which, he believed, had originated in a nerve loop. This view is strengthened by the fact that fully half of the loops show neuroma-like enlargements at their apexes. Axenfeld,[5] who first described these loops from microscopic preparations, later was the first to report a case observed clinically. The patient complained of intermittent pain from

2. Fuchs: Klin. Monatsbl. f. Augenh. **60**:3, 1918.

3. Fischer: Ztschr. f. Augenh. **66**:59, 1928.

4. Kyrieleis: Arch. f. Ophth. **119**:119, 1927.

5. Axenfeld: Heidelberg, Kongressber., 1895, p. 122; Cong. internat. d'opht. d'Utrecht, Compt. rend., 1899, Amst. **9**:162, 1900. Axenfeld and Naito: Heidelberg, Kongressber., 1902, p. 134. Axenfeld: Klin. Monatsbl. f. Augenh. **75**:602, 1925.

the movement of the lower lid over the eyeball. Examination revealed below in the vertical meridian, 3 mm. from the limbus, a sharply circumscribed and demarcated grayish-white flat prominence, 2.5 mm. in diameter. It was free from any signs of irritation or inflammation, and the conjunctiva moved freely over its surface. The temporal half of this mass joined the sclera, while the nasal end could be moved freely, and under it lay a dark emissary through which the adherent temporal portion seemed to emerge. The mass was sensitive to the touch, and extreme pain was elicited by pulling the free nasal end. Excision showed it to be composed of nerve tissue. It left a hypesthesia of the cornea almost to the horizontal meridian. Kyrieleis also produced hypesthesia by the excision of a loop.

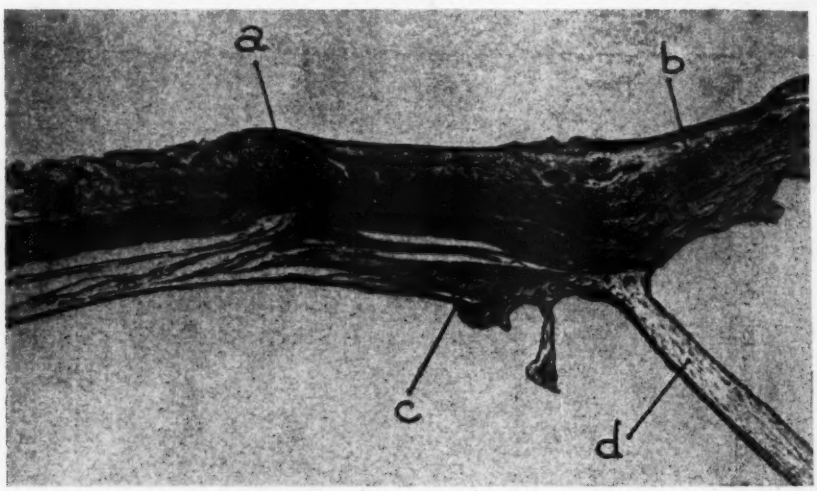

Fig. 3.—A nerve loop extending to the external scleral surface: *a*, the cupola of a retroverted nerve loop lying on the external scleral surface and covered only by conjunctiva; the scleral emissary through which it extended is somewhat dilated and ectatic owing to the fact that this was a glaucomatous eye; some of the ciliary muscle is pushed into the foramen; *b*, the cornea; *c*, the ciliary body, and *d*, the iris with peripheral anterior synechiae.

The embryology of the intrascleral nerve loop is in dispute. Axenfeld thought that it probably served as an anchorage in the sclera at the point where the nerve changes its meridional direction for a circular one. Groenouw [6] thought that the artery that often accompanies the loop coursed from within out, and that this artery pulled the already formed nerve with it to a greater or lesser degree. The objections to this are that the loop is usually not accompanied by an artery, and that when it is, the artery is in a majority of cases unmistakably an anterior ciliary artery with a course from without in. Fritz assumed the artery

6. Groenouw: Klin. Monatsbl. f. Augenh. **43**:637, 1905.

to be an anterior ciliary one, and claimed that as it courses inward through the sclera it is growing in a direction opposite to the ciliary nerve, thereby serving as a hindrance to the forward course of the nerve. The nerve therefore reacts by growing in loop formation around the obstacle. The objections to this explanation are that the nerve loop and anterior ciliary artery do not always share the same emissary, and also it is probable that if the nerve looped to avoid an obstacle it would do so along the course of least resistance, which would be in the more rarefied subchoroidal space or choroidal tissue instead of in the denser scleral tissue. Fischer studied human embryos in all stages of develop-

Fig. 4.—A nerve loop extending through the sclera to the external surface: *K*, the cupola of the loop with a neuroma-like enlargement; *S*, ascending stem of the nerve; *S'*, descending stem of the nerve; *P*, space between the ciliary body and sclera (reproduced from Fischer: Ztschr. f. Augenh., 1928, vol. 66).

ment, and concluded that the ciliary nerve reaches the region of the ciliary body before the sclera and uveal layer have differentiated. This greater capacity for growth, and therefore the greater length of the nerve, is compensated for by a loop or fold brought about by a shifting of the sclera and ciliary body in the act of development and differentiation. This explanation has much to recommend it.

E. Fuchs reported that from a study of human embryos he did not think that the correct explanation of these loops rests on a mechanical basis, but rather on an abnormal tendency to growth. The ciliary nerves

grow more rapidly than the sclera, and their greater length is compensated for by their duplication or folds in the sclera. He cited a similar abnormal growth of the optic nerve whereby aberrant bundles of the nerve grow blindly into the sheaths and lose themselves there. Also similar abnormal growths of the optic chiasm are seen in which a conical process composed of loops of aberrant nerve bundles extend out from the anterior wall. Thus opinion is divided between the theory that these intrascleral nerve loops are a normal anatomic variation and the theory that they are an abnormal tendency to growth. There are several factors in favor of the latter:

1. About 65 per cent of the cases show a neuroma-like enlargement of the cupola.

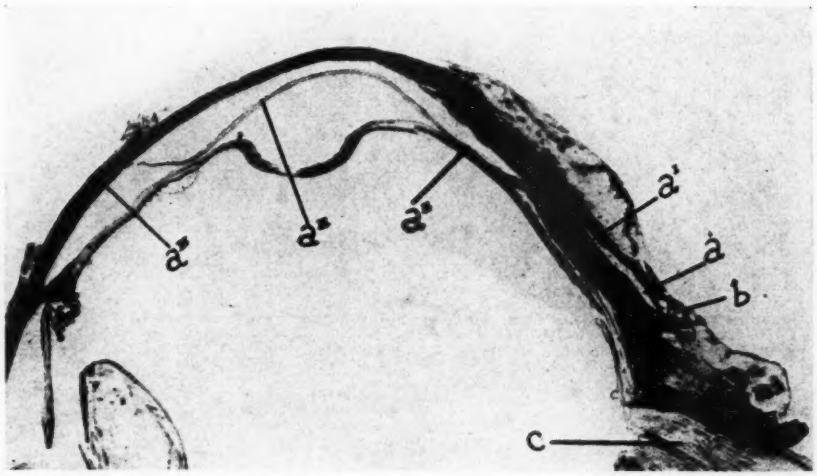

Fig. 5.—Section showing the long ciliary nerve throughout its entire course: *a*, the long ciliary nerve before entering the sclera posteriorly; a^{I}, coursing through its emissary in the sclera; a^{II}, a^{III}, in the subchoroidal space; a^{IV}, looping obtusely in the sclera before entering the ciliary body; *b*, short ciliary nerves cut in cross-section; *c*, the optic nerve.

2. A neuroma has been reported as arising in all probability from one of these loops.

3. A loop has been reported in an eye with a congenital coloboma of the iris.

4. Similar abnormal growths are sometimes seen in the optic nerve and chiasm.

The clinical characteristics of an intrascleral nerve loop which serve to make the diagnosis are as follows: It is a grayish-white nodule from one to several millimeters in diameter (fig. 1). The cupola of the loop may just peep through the external surface of the sclera, in which case it will be just visible and practically flush with the scleral surface. All

degrees from this slight manifestation to several millimeters of elevation may be seen. The loop may be flexed on itself or may show a mushroom-like shape due to enlargement of its cupola. On lifting the loop away from the sclera or pushing it to one side, it is sometimes possible to see that it emerges from a scleral emissary, the orifice of which may show a slight amount of pigment. A small cyst may accompany the loop. The loop is invariably 3 or 4 mm. from the limbus, usually on the nasal side, though it is found inferiorly also and occasionally superiorly and temporally. The conjunctiva moves freely over its surface. Even after the instillation of cocaine the loop is tender to touch, and pulling or rubbing it causes pain. It gives rise to no symptoms except that in rare cases when it is very large the patient may be conscious of the lid rubbing over the eyeball, as in the case reported by Axenfeld. Excision results in hypesthesia of the cornea over a sector corresponding to the site of the loop, but there is no indication that the innervation of the iris and ciliary body is affected.

When the diagnosis of a loop is made, it is obvious that no surgical or other treatment is indicated. I know of two instances in which a loop was excised in the belief that it was a small new growth, and still another instance in which excision was advised. These cases are cited as evidence that the recognition of an intrascleral nerve loop is of some clinical importance. The slight grayish color lends a suggestion of pigment.

This presentation is based on the study of four microscopic specimens and seven clinical cases, in all of which the loop extended to the external scleral surface. Microscopic specimens in which the loop extends partly through the sclera and clinical cases in which it just peeps through the external scleral surface are not at all uncommon. Indeed, if a point is made of looking for these loops, it is surprising how often they may be seen.

EPIPAPILLARY TISSUES*

BERNARD SAMUELS, M.D.
NEW YORK

The material on which this paper is based consisted of microscopic preparations from 300 eyes. Unless otherwise stated, the eyes were those of adults. In seventy of the slides the tissues were thought to have been present at birth, while in the remaining number they were considered the outcome of disease. According to their origin they are divided into the following groups:

1. CONGENITAL TISSUES

A. Remains of the Hyaloid Artery. These consist of: 1. Strands of varying length and thickness, of which there were eighteen examples. Fifteen came off from the nasal crest of the pit, in the horizontal meridian, and only three from the bottom of the pit. 2. Clumps of pure glial cells, representing a more advanced stage in the involution of the artery than the strands. There were seven, all situated on the nasal crest, in the horizontal meridian. 3. Membranes of pure glial cells, spread over the medial aspect of the nerve head. Glial membranes were more frequently found in this survey than any other vestiges of the hyaloid artery. They were encountered in fully three-fourths of the specimens ascribed to congenital origin. They were sometimes accompanied by a strand or a clump, but many times they were found alone. 4. Cellular masses in the pit (the so-called central connective tissue meniscus of Kuhnt[1]). The peculiar collection of cells that is often seen in the depth of the pit is frequently found alone, but a strand, clump or membrane may be present.

B. Voluminous Connective Tissue in the Pit. There were five examples in which the entire pit was filled by a mass of dense connective tissue, in the midst of which the retinal vessels were encased.

C. Tissue in Connection with Other Intra-Ocular Anomalies. In the entire collection there was but one instance of this. A delicate strand of connective tissue, all that was left from the wall of a hyaloid artery, projected from the nasal side of a nerve, and in the nearby retina there was an area of medullated nerve fibers. It is worthy of note that when strands are found, the nasal side of the nerve is apt

* Submitted for publication, June 6, 1931.
* Presented at a meeting of the American Ophthalmological Society, June 2, 1931.

1. Kuhnt: Arch. f. Ophth., 1897, p. 25.

Fig. 1.—Tuft of pure glial cells that springs from the margin of a glial membrane on the nasal surface of the optic nerve (congenital).

Fig. 2.—Remains of the wall of a hyaloid artery, denuded of glial covering and showing compressed endothelium in the lumen (congenital).

Fig. 3.—Typical glial membrane (congenital).

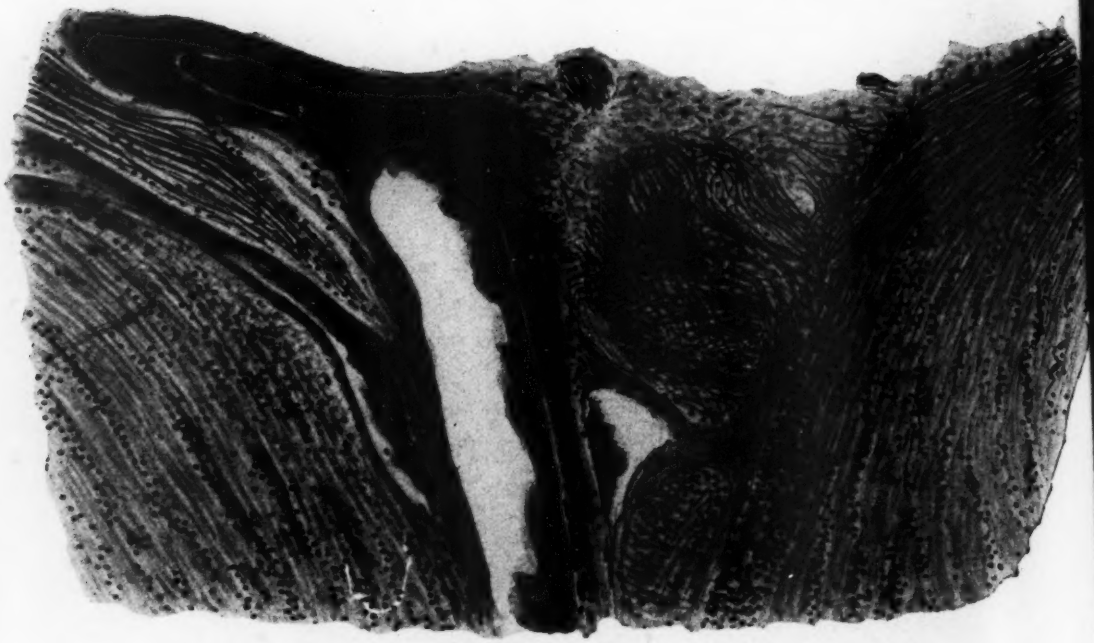

Fig. 4.—Admixture of neuroglia and connective tissue in the pit (congenital).

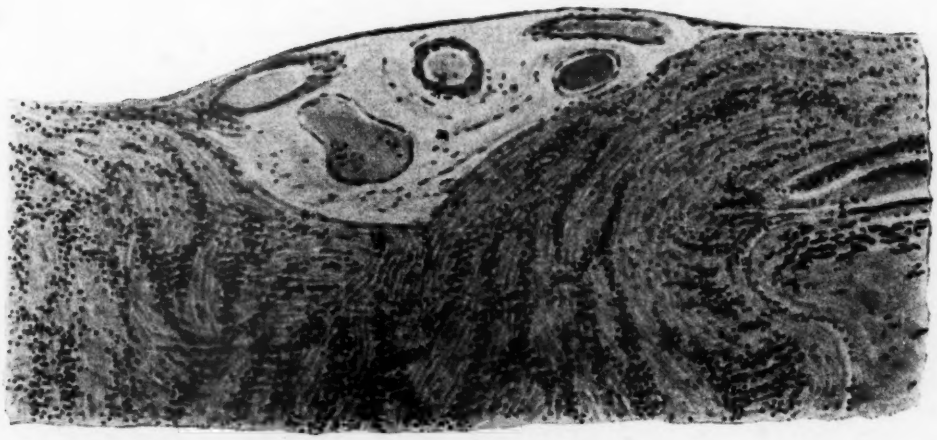

Fig. 5.—Cross-section of retinal vessels encased in a mass of dense connective tissue covered by a glial membrane (congenital).

Fig. 6.—Group of glial cells suspended in the vitreous by delicate strands from a glial membrane (congenital).

to be unusually prominent, and in these eyes the retinal vessels are very superficial, being in contact with the nerve fiber layers only on the posterior surface. As they spread out, the vessels dip deeper into the retina.

2. ACQUIRED TISSUES

At the papilla there are two elements of very different origin that have the property of proliferating when irritated, namely, the con-

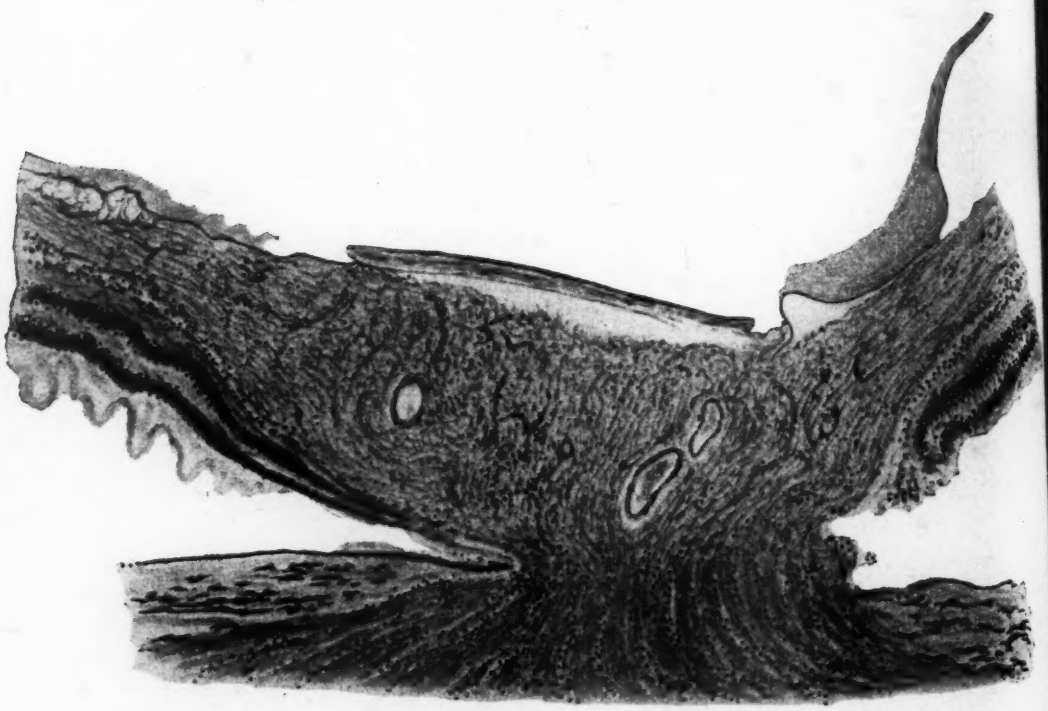

Fig. 7.—Glial membrane detached by an albuminous fluid (congenital). The fellow eye shows a similar condition.

nective tissue of the blood vessels and the neuroglia. They may proliferate together or separately, so that in the first instance there would result a mixed tissue, and in the second instance a tissue of pure neuroglia or one of pure connective tissue. Fuchs [2] demonstrated that the connective tissue septums of the optic nerve, including the central vascular strand and the cribriform fascia, contain a considerable admixture of neuroglia in the layers facing the nerve bundles. As the vessels of the retina emerge from the pit they carry with them in their adventitia this intermingling of connective tissue and neuroglia. A similar

2. Fuchs: Arch. f. Ophth., 1916, vol. 91.

admixture of the two tissues may be found at times in the meniscus at the pit. Acquired tissues are accordingly classified as follows:

1. Neuroglia alone. This originates from the proliferation of the neuroglia in the anterior layers of the nerve. Glial cells remaining from the involution of the hyaloid artery may also proliferate.

2. Connective tissue alone. The sole source of this is the walls of the vascular system.

3. Mixed tissue. A simultaneous growth of connective tissue and neuroglia is very frequent.

Fig. 8.—Remnant of the hyaloid artery and medullated nerve fibers in the retina (congenital).

4. Tissue derived from the extension of abnormal tissue from the surface of the retina.

5. Tissue acquired by traction. The adjoining retina may be drawn into a pathologic excavation with the recession of the cribriform fascia.

HISTOLOGY OF THE TISSUES ON THE NERVE

Congenital Tissues.—1. Strands: Swollen, hyalinized walls characterize these. In two cases the walls were so swollen as to give a flask-like shape to the strands. The distal ends of the strands may be smooth and round or frayed out, and at times they taper to a fine point. In

several specimens the glial covering had entirely disappeared from the walls. There were cases in which the lumen is indicated by compressed endothelial cells. In only two cases blood cells were found in the lumen. The occlusion of the lumen appears to be an early step in the involution of the artery. The proximal ends of the strands are inserted superficially in the nerve fiber layers. More than once in this series the inser-

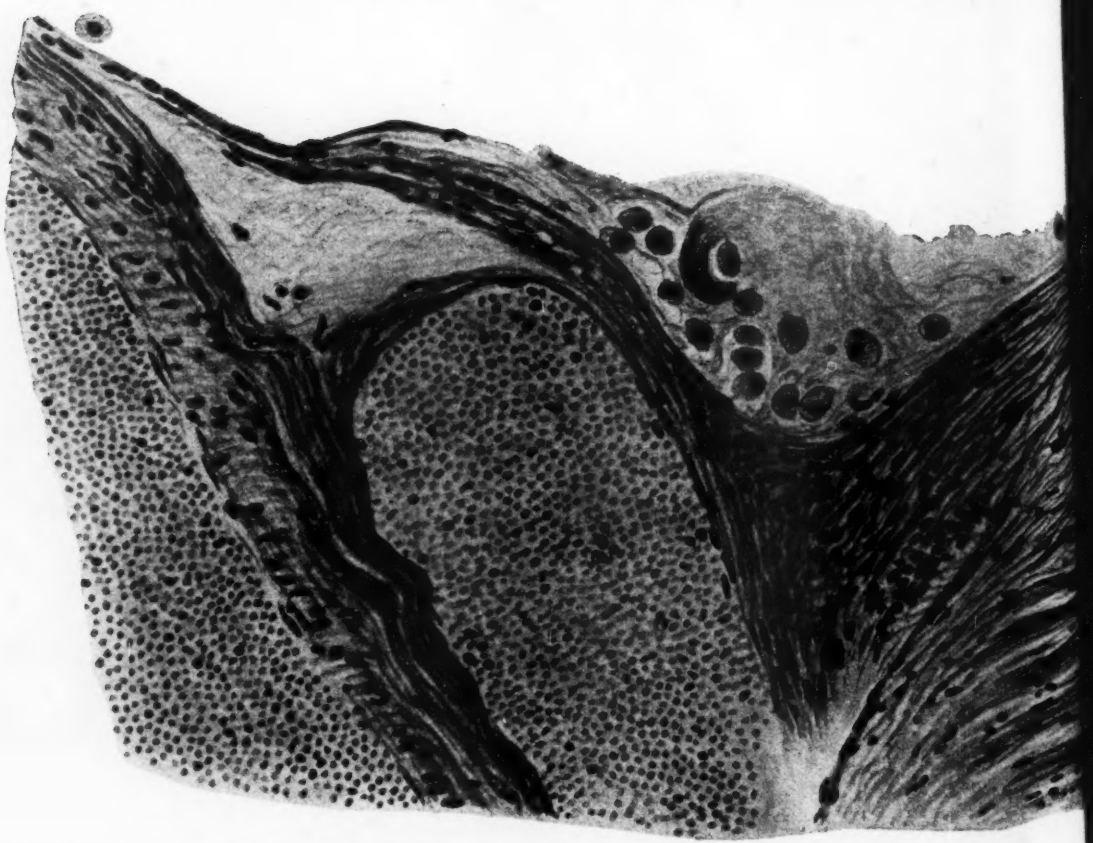

Fig. 9.—Proliferation of pure glial tissue caused by irritation from the presence of a piece of steel in the vitreous. Note the detachment of the glial membrane by an albuminous fluid containing a few cells.

tion was at some distance from any retinal vessel. One specimen was of particular interest, in which a thick strand projected from the bottom of a glaucomatous excavation. The elastic fibers of the intima are well preserved, showing as fine longitudinal lines. The glial mantle is preserved, passing off on either side into a thick layer of newly proliferated neuroglia that covers the floor of the excavation.

2. Clumps of Pure Glial Cells. They are apt to be of small extent and to be low and round, with smooth surfaces. The cells are elongated.

Exceptionally, instead of clumps, there are bristly tufts of angular glial cells, suggesting a chestnut bur. Whenever a strand or clump springs from a glial membrane, it is generally found at the nasal fringe of the membrane.

3. Membranes of Pure Glial Cells. These cells are more elongated than those of the clumps, so that they resemble endothelial cells. There may be one layer of cells in one area of a membrane, and elsewhere there may be several layers. Membranes imperceptibly taper off into the limitans interna of the retina on the medial side. On the lateral side they may reach to the pit, beyond which they seldom go. In the absence of a meniscus, a memrane at the pit lies in direct contact with the

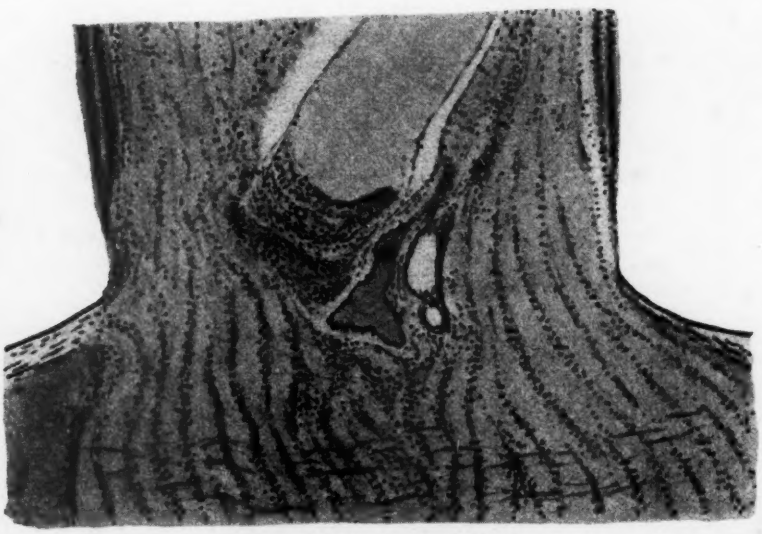

Fig. 10.—Massive proliferation of neuroglia, with a few connective tissue fibers, in a case of retinoblastoma, probably the result of irritation.

nerve fiber layers and with the walls of the vessels. The membranes separate readily from the surface, judging by their tendency to do so as an artefact. Four eyes showed glial membranes separated from the surface by a slightly albuminous fluid containing a few cells. In one case the separation was bilateral. In detaching, a glial membrane may take away with it the contiguous limitans interna of the retina. In this way cystic spaces at the papilla may extend beyond the margins of the nerve.

4. Cellular Masses of the Pit. These are loosely attached to the nerve, being anchored by delicate neuroglia strands or fine fibers of connective tissue. When no glial membrane is present, the vitreous surface is irregularly defined. Capillaries in the meniscus were not encountered in any of the sections.

5. Voluminous Connective Tissue in the Pit. This is very dense and homogeneous, containing few nuclei and no capillaries. It may be considered as a hypertrophy and coalescence of the walls of the vessels at the nerve head. The thick tissue stands out in sharp contrast to the frail

Fig. 11.—Proliferation of connective tissue and neuroglia produced by toxins from iridocyclitis.

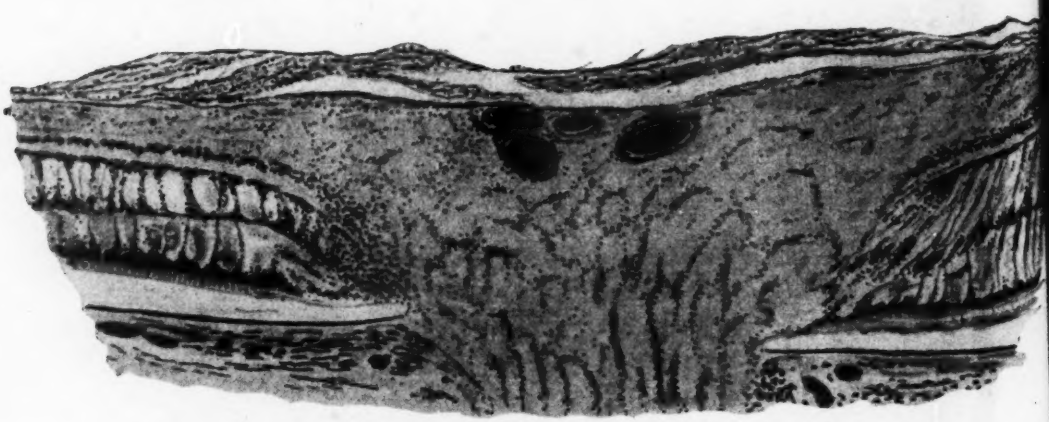

Fig. 12.—Delicate membrane of new-formed connective tissue, lying lightly on the surface.

nerve fibers and their columns of nuclei. Extensions of the central plug may cover the papilla in all directions. Such extensions show no preference for the nasal side, in contradistinction to glial membranes, which are confined to the nasal side. In the specimens examined there were no prolongations of the congenital connective tissue into the vitreous.

Acquired Tissues.—1. Neuroglia: Recently proliferated glial cells are oval or round, and larger than normal glial cells. The nuclei take the stain lightly, while the protoplasm stains heavily, in consequence of which a new growth of glial cells has a luxuriant appearance under the

Fig. 13.—Connective tissue membrane showing early hyalinization (acquired).

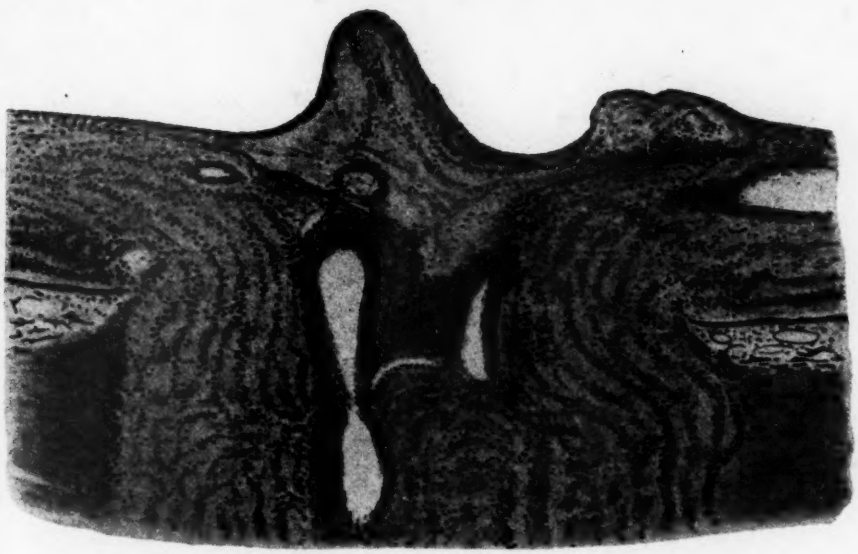

Fig. 14.—Complete hyalinization of connective tissue that has been thrown into folds (acquired).

microscope. A proliferation of glial cells is a frequent occurrence in old iridocyclitis of mild degree, no doubt caused by toxins that have found their way backward. A proliferation of glial cells is very characteristic of many deep glaucomatous excavations. Here the reason is impaired

circulation and the disappearance of the nerve fibers. In the brain the neuroglia fills out gaps produced by death of nerve fibers, and at the papilla it does the same thing. There are specimens in which congenital neuroglia membranes are thickened by proliferation. Now and then glial cells in the anterior layers of the nerve break through the surface at points and produce nodular formations. These may detach spontaneously and become free in the vitreous as "cellular spheres."

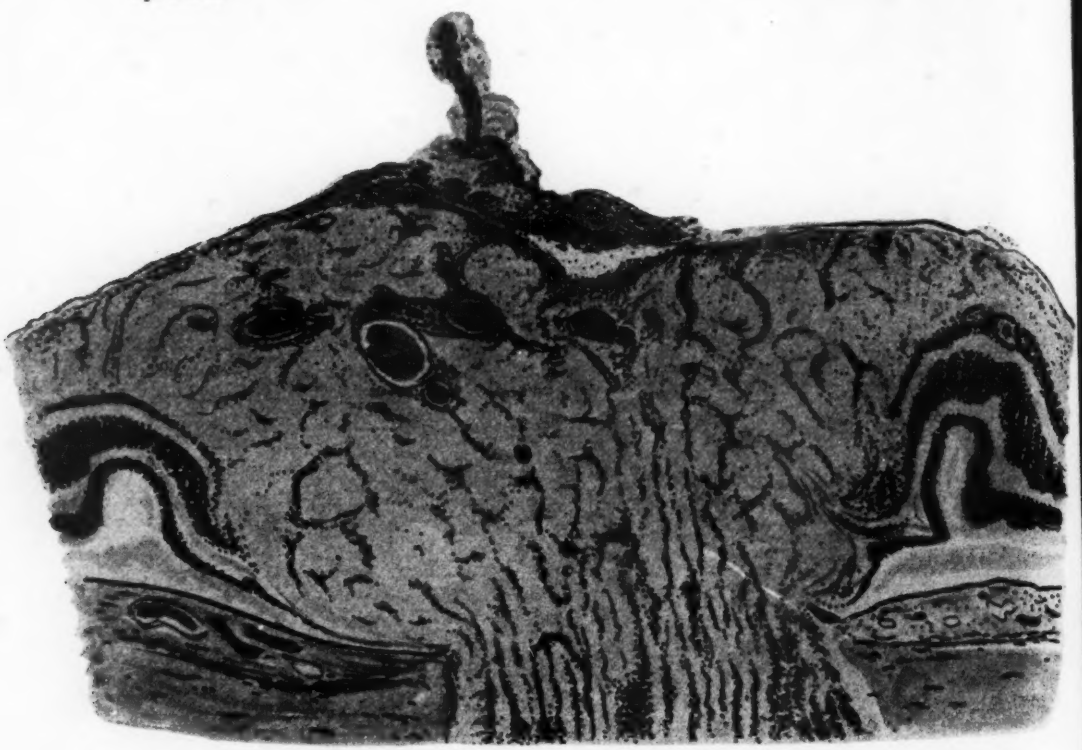

Fig. 15.—Formation of a cord of connective tissue on the surface of an edematous nerve, in the early stages.

2. Connective Tissue: (*a*) Membranes. Exudates on the nerve head organize most frequently in the form of membranes. The connective tissue is stimulated to proliferate under the influence of toxins of severer intensity than those causing a growth of the neuroglia alone. Connective tissue membranes are very lightly attached to the surface so that they separate easily. They may be lifted up by serum, blood or lymphocytes collecting on the under surface. Many of them pull away by their own contraction. These membranes may appear lamellated, due to the organization of repeated hemorrhages or exudates on the posterior surface. Acquired membranes may confine them-

selves to the nerve head, but generally they extend beyond. On the retina they lie still more lightly than on the papilla, because of the smooth, homogeneous limitans interna. They frequently break away completely from the retina, while adhering to the nerve from which they

Fig. 16.—Cord of connective tissue projecting in the path of Cloquet's canal (acquired).

wave in the vitreous. Some membranes are rich in blood vessels; others contain no blood vessels. It seems that all connective tissue formations that are acquired sooner or later undergo hyalinization. One part of a membrane may be cellular and progressing, while another part may

Fig. 17.—A very vascular layer bridges over a pathologic excavation that is partially filled by a proliferation of neuroglia.

Fig. 18.—Voluminous mass of hyalinized tissue at the bottom of a deep excavation.

be thrown into hyalinized folds containing no nuclei. Great hyalinized plugs may partially fill out the deepest excavations. In one of the preparations the connective tissue along the walls of the excavation has been transformed into bone. Because of the bleeding that accompanies the laying down of many membranes, it happens that the nerve may be sealed off by a condensed membrane permeated with blood pigment.

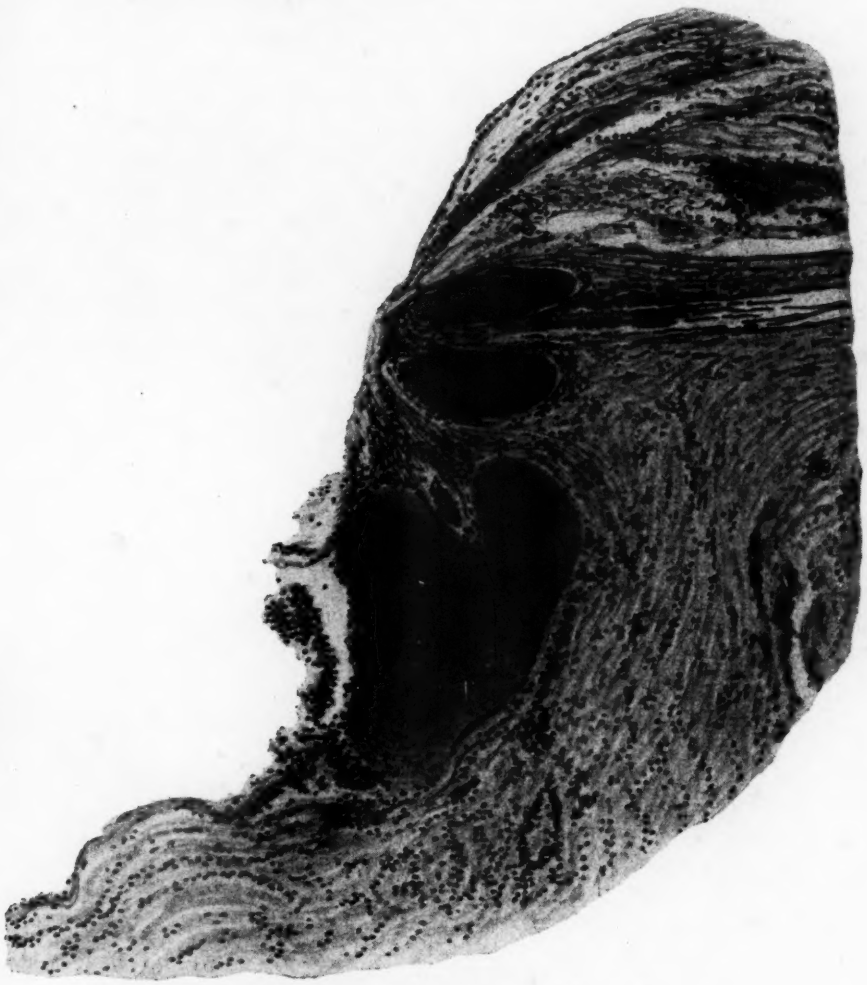

Fig. 19.—Bone formation on the side of an excavation.

Membranous formations in glaucomatous excavations have a pronounced tendency to detach. As the recession of the cribriform fascia increases, the overlying tissue holds back, probably by its own contraction. In pulling away from the surface of the cribriform fascia, there is brought about a rarefaction of the deeper layers, so that in the sections a characteristic appearance is produced. The layer facing the

vitreous is composed of a row or more of small, elongated cells, suggesting endothelium. Below this is a thicker layer, containing many lymphocytes and adhering closely to the first layer. A loose scaffolding of tissue makes up the posterior layers, the elements of which are drawn out in an anteroposterior direction. These layers are apt to contain cystic spaces. At a later stage, owing to a rupture of the anterior layers, the entire formation may fall back against the walls of the

Fig. 20.—Membrane of connective tissue creeping over the margin of the nerve from an inflammatory focus on the retina.

excavation. By compression they would be reduced to a homogeneous layer lining the excavation. In this manner some of the deepest excavations present dense smooth surfaces, while other excavations not so old and deep may have ragged surfaces.

(*b*) Cordlike Structures. In some preparations, particularly in papilledema, the exudate or hemorrhage, instead of being distributed evenly over the surface, is piled up in the pit in the shape of a cone, with the apex forward. Such an observation suggests that there is a

potential space in front of the nerve, corresponding to Cloquet's canal, in which serum and cells may accumulate, and in which connective tissue may form. The apex of a cone of connective tissue may elongate into a cord, which may move to and fro in the vitreous, or it may proceed forward to be attached to a cyclitic membrane. In the several "cords" studied, no blood vessels were seen, in which respect they resembled membranes. The cells of the cords were drawn out parallel to the surface. Cords produce traction on the nerve, but they do not directly cause detachment of the retina, being confined to the central area of the papilla.

Nodules of connective tissue, similar in shape to those of neuroglia, were seen on the surface in a few specimens. They may also detach, being the source of "fibrous spheres" that circulate in the vitreous.

(*c*) Proliferation of Mixed Origin. A typical picture is that of neuroglia in the form of folded strands or membranes lying in the midst of new connective tissue. In some examples, the two tissues, while proliferating at the same time, were separated from each other. Thus, in a glaucomatous excavation of a highly myopic eye, a mass of glial cells was present on the nasal side, under the scleral spur, which is a favorite site for the growth of neuroglia, and widely removed there was a mass of connective tissue on the temporal side. Where the two tissues intermingle, in the end, the connective tissue alone will survive; the neuroglia, being more delicate, appears to be choked out.

A remarkable case was that of an infant's eye that was excised because of early glioma exophytum (retinoblastoma) in the anterior part. The entire nerve and the adjacent retina were clothed with a mass of glial cells, showing a few connective tissue fibers. Probably this was a mixed proliferation caused by the irritation of toxins emanating from the tumor.

Another unusual case of mixed proliferation was that of an eye that was injured ten years previous to enucleation. The deep excavation was occupied by a quantity of connective tissue and neuroglia, containing a few blood vessels. From this mass there extended over the margins of the nerve a thick membrane, which gradually became thinner as it adhered to the surface of a detached retina almost to the ora serrata. No other membrane was traceable for such a great distance from the nerve. In the immediate neighborhood of the nerve the membrane retained its mixed nature, but as the distance increased and as it thinned out, it took on the appearance of a pure neuroglia membrane.

(*d*) Tissue Extending over the Nerve from the Surface of the Retina. In two preparations of retinitis proliferans there were connective tissue membranes on the retina from which strands projected

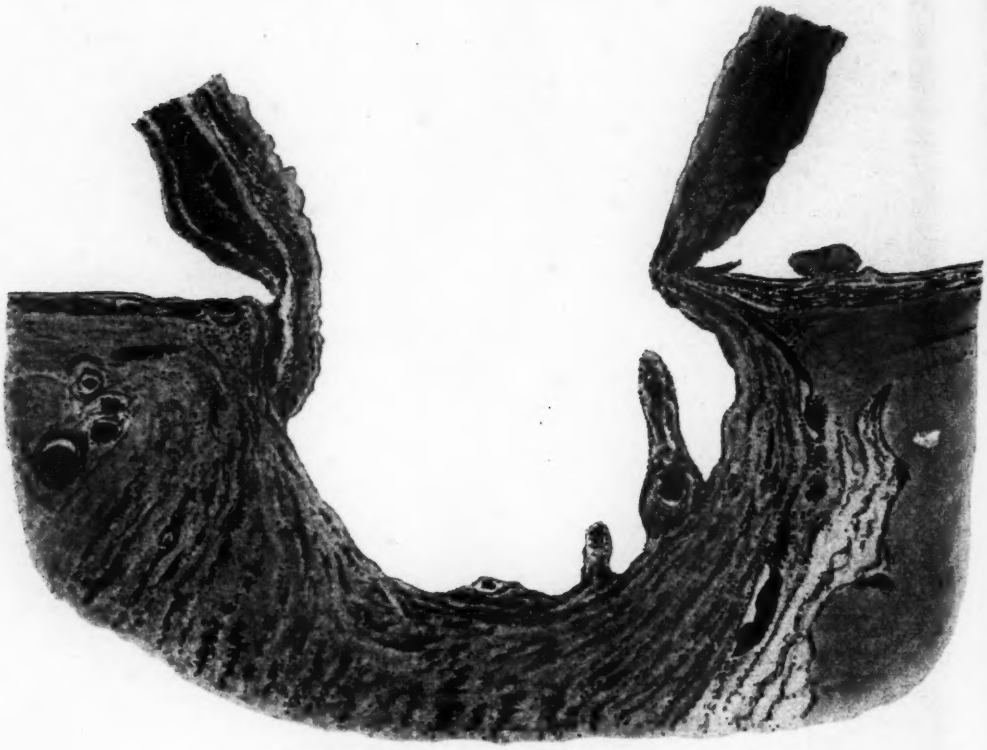

Fig. 21.—Retina drawn into the excavation by traction.

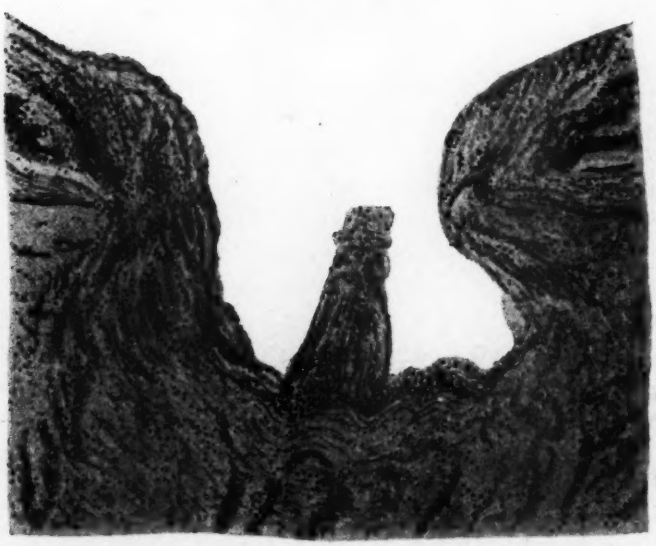

Fig. 22.—Remains of a hyaloid artery at the bottom of a glaucomatous excavation, with a proliferation of glial cells on the adjoining surface.

into the vitreous. The strands were situated at some distance from the nerves, which were excavated. In one case the membrane bridged over the excavation like a roof, and in the other case it clung to the walls of the excavation.

(*e*) Tissue Acquired by Traction. There were instances of congenital strands and membranes on the medial side that had been drawn into excavations with the retina. The portion of the retina that had glided over the scleral spur was thin, because not only was there the usual atrophy of the nerve fibers seen in glaucoma, but there was also a disappearance of the rods and cones, with a thinning or absence of the external nuclear layer.

Site of Predilection for All Types of Tissues.—Whether of congenital or acquired origin, tissues are more likely to be found on the nasal surface of the papilla. It is here that congenital structures have their seat in the remnants of the hyaloid artery, and acquired tissues develop more readily on this side because of the richer blood supply and the greater abundance of the neuroglia between the more numerous nerve bundles.

CORRELATION OF HISTOLOGIC OBSERVATIONS WITH OPHTHALMOSCOPIC PICTURES

Congenital Tissues. 1. Remains of the Hyaloid Artery. It is assumed that the glial mantle is possessed of a transparency similar to that of the neuroglia in the retina, but whether or not the connective tissue of the walls is transparent depends on the thickness and on the degree of hyalinization. The element of thickness plays an important rôle in the transparency of all tissues. Thus thin connective tissue may be transparent, while thick neuroglia may be quite opaque.

2. Clumps of Glial Cells. Because of their thickness and being very cellular, they should reflect the light, but probably most of them are too minute to be seen with the ophthalmoscope. However, bristly clumps of glial cells are most likely the cause of the tiny glistening elevation on the nasal crest that is occasionally observed in the fundi of young persons.

3. Membranes. The thin veils that slightly obscure the blood vessels on the disk, as well as the nasal border, are congenital membranes of pure neuroglia. It is probable that only the thicker membranes are visible. The frail, translucent walls that outline bullous formations at the disk are glial membranes enclosing a clear fluid that may contain some cells. Transparent pedicles that move to and fro from the nerve may once have been the walls of cystic spaces that have ruptured, or membranes that have floated off from the surface.

4. Cellular Masses in the Pit. These are observed clinically because, being very cellular, when they are heaped up they reflect the light.

They appear as bluish, wooly tissue in the depths of the pit into which the vessels vanish. Occasionally, minute translucent bodies are seen suspended in the vitreous in front of the nerve. They are collections of glial cells that have gotten loose from the nerve but to the nasal side of which they are still anchored by invisible threads of neuroglia.

It is instructive to watch with the ophthalmoscope the excursions of cellular masses in the pit and of nearby glial membranes produced by the pulsations of the blood vessels.

5. Voluminous Connective Tissue in the Pit. This accounts for the pure white area in the center of the nerve, usually on a level with the retina, that is typical of many hyperopic eyes. Winglike extensions from such a central mass of connective tissues may screen almost the entire nerve from view.

Acquired Tissue.—Many of the pathologic structures that are revealed under the microscope are not seen clinically, because they occur in the eyes that are too severely damaged to be examined with the ophthalmoscope.

1. Neuroglia. Verhoeff [3] stated that the waxy appearance of the disk in retinitis pigmentosa is due to gliosis. In a similar way, the waxy, homogenous appearance of the depths of some glaucomatous excavations is probably the effect produced by a thick layer of neuroglia that obscures the perforations in the cribriform fascia.

2. Connective Tissue. Hyalinized portions of membranes have in all probability a degree of transparency analogous to that of the so-called colloid bodies on the choroid. The latter, having little or no pigment on the anterior surface, allow the rays of light to pass through to the choriocapillaris, giving the yellow color associated clinically with these glistening bodies. On the nerve head hyalinized tissue, while it may glisten, appears white because of the white background. Congeries of blood vessels, noted clinically in far advanced cases of glaucoma that were almost on a level with the retina, were portrayed in the sections as vascular membranes, some of which concealed deep excavations.

COMMENT

Congenital Tissues.—1. Strands at the nerve head, to be of congenital origin, should be traceable to the nasal side in the horizontal meridian. It is exceptional that they spring from the bottom of the pit, and it appears that they are never on the temporal side of the papilla. 2. All delicate, transparent veils on the nasal side of the nerve are of pure glial origin, and should be referred to as neuroglia membranes. The walls of cystic spaces in front of the nerve are of the

3. Verhoeff: Tr. Am. Ophth. Soc. **28**:176, 1930.

same origin. 3. As the mass of cells at the bottom of the pit may contain both connective tissue and neuroglia a noncommittal term would be simply "central meniscus." 4. The pure white area observed in the center of many nerve heads represents the only connective tissue ever present of congenital origin, according to the survey, aside from the usual walls of the vessels. Any congenital connective tissue membranes are extensions from such a mass in the pit. 5. So far as glial membranes are concerned, they cover the medial aspect of the nerve, so that the lateral half is the part that is always free from a membrane.

Acquired Tissues.—1. A proliferation of pure glial cells is more apt to be found on the papilla in glaucomatous excavations than in any other condition. 2. In eyes that have been inflamed, the usual strands and membranes seen have no connection with any remnants of the hyaloid artery. Unless tissues in diseased eyes accurately fulfil the typical requirements of congenital tissues in the way of location, extent and shape, they are most likely of acquired origin.

Finally, it is possible to distinguish clinically, with a fair measure of certainty, the various types of congenital tissues, and to differentiate them from the various types of acquired tissues.

CONGENITAL ABSENCE OF THE LACRIMAL PUNCTA AND OF THE CANALICULI*

EVERETT L. GOAR, M.D.
HOUSTON, TEXAS

My interest in this rare condition was stimulated by a patient who was first examined about two years ago. As reports of successful operative results are seldom found in the literature, this account may interest the Society. The condition was encountered but once in Mooren's series (cited by Burnett [1]) of 108,416 examinations. Burnett also quoted von Walther as saying that "atresia of the puncta and the beginning of the canals, aside from monopsia and micropsia is not to be met with as a congenital defect." This statement is, of course, incorrect unless one agrees that the condition is the result of prenatal or postnatal inflammation. In an incomplete review of the literature, I have found twenty-two cases of absence of the canaliculi. I have not attempted to differentiate between absence and atresia in this review, as it is not important to do so. In some of the reports, it was impossible to distinguish whether the puncta were absent or imperforate, and also whether the canaliculi were present.

REPORT OF A CASE

Miss R. W., aged 22, a stenographer, consulted me on Sept. 28, 1929, complaining of watering of the left eye, which she had first noticed two years before. There had been no inflammation of the eyes. She had a small error of refraction for which she had worn the proper correction for several months; however, epiphora continued and was constant and annoying. Examination revealed that the puncta and papillae of the left lower and upper lids were completely missing. The points where they should have been were entirely smooth, with no elevation, depression or discoloration. I dissected the lower margin of the lid for about 6 mm., but could find no trace of a canaliculus, so did not search for the upper.

Operation was performed on October 15. A needle was passed through the semilunar fold into the region of the lacrimal sac, and fluid was injected. The solution came promptly into the nose, showing that a sac was present and that the duct was patent. After infiltration with 2 per cent procaine hydrochloride, a vertical incision was made through the semilunar fold behind the caruncle, and the dissection was carried through the lateral wall of the sac. A large probe was passed through the opening into the nose. Two days later a metal style, with the head bent at right angles to the shaft, was introduced. The style was loosened every few days and was left in place for ten weeks. After removal of the style,

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* Read at a Meeting of the American Ophthalmological Society, Asheville, N. C., June 2, 1931.

1. Burnett: Arch. Ophth. **13**:53, 1884.

granulations that appeared at the orifice were destroyed two or three times with the cautery. Large Bowman probes were passed twice a week for a month; then the intervals between probings were gradually lengthened.

The patient reports about once in three months for observation. A no. 8 Bowman probe slips readily into the orifice, which is in a smooth funnel-shaped depression immediately behind the caruncle. Fluids instilled into the eye pass readily into the nose. There is no more epiphora. The artificial canal has apparently epithelialized; after nearly two years it is justifiable to report a cure.

REVIEW OF THE LITERATURE

The most concise description of the development of the lacrimal apparatus that I have seen is that of Whitnall.[2] He stated essentially as follows:

Fig. 1.—Appearance of the patient, with a probe through the opening.

The lacrimal passages are developed along the line of the cleft between the lateral nasal and maxillary processes of the embryonic face: they are formed by the canalization of a solid rod of ectodermal cells formed beneath the surface along the line of the cleft. The first appearance of the rod is at a point corresponding to the medial part of the conjunctiva of the lower lid. The lower end elongates and sinks beneath the surface, the whole rod following and becoming surrounded by mesenchymal tissue, but retaining its contact with the skin longest at the point of origin. The first appearance has been seen in an embryo of 9.5 mm. in length, and the rod was found completely separated from the surface in one of 15 mm. The canaliculi are formed secondarily by budding from the upper end of the rod and reach the surface of the lid margins about the same time that the lower end of the rod reaches the nasal cavity. The inferior canaliculus appears to be the direct continuation of the main stem, the upper one being a secondary outgrowth.

2. Whitnall, S. E.: The Anatomy of the Human Orbit and Accessory Organs of Vision, London, Oxford University Press, 1921, p. 245.

The inferior canaliculus in its growth upward cuts off a part of the lid margin which is destined to form the lacrimal caruncle. The process of canalization begins by disintegration of the central cells, first in the middle of the lower canaliculus, then in the upper and last in the lacrimal sac. The puncta and the inferior orifice of the duct are the last to open; the former open just before the eyelids separate about the seventh month; the latter remain closed until the end of the eighth month or even until after birth.

It seems probable that absence of the puncta and canaliculi is due to the failure of the cells in the epithelial rod to disintegrate, for if it were due to failure of this rod to form and grow upward, the caruncle would also be absent. In those cases in which the lacrimal sac is present—as in the one I have recorded, and all the reports I have

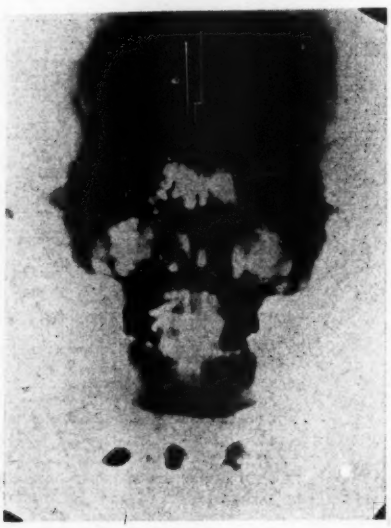

Fig. 2.—X-ray film taken after the injection of iodized poppy seed oil 40 per cent.

reviewed—the canalization proceeds normally in the lower part of the epithelial rod. In patients with atresia of the puncta in whom the papillae and canaliculi are present, atrophy of the epithelial cells has occurred to the point of perforation and then ceased, leaving the thin conjunctival membrane to remain intact.

Desmarres,[3] in a second edition of his textbook (1854), under the caption "Congenital Absence of the Puncta and Canaliculi," stated that the condition is far from rare, and that many authors have noted a goodly number of patients with lacrimation in whom the canaliculi were lacking. There was no evidence of inflammation, but all of the patients

3. Desmarres: | Traité théorique et pratique des maladies des yeux, ed. 2, Paris, 1854, vol. 1, p. 280.

had had conjunctival infections in infancy which had continued for some time. He did not know whether the absence was congenital and accounted for the ocular disorder, or whether the canaliculi were closed during the progress of early inflammation.

In 1846, Blanchet [4] presented a case before the Royal Academy, that of a man aged 25, in which all four puncta and part of the canaliculi were missing. He had succeeded in opening one punctum and one canaliculus on the right, but did not give the technic. He presented the case in order to have the preoperative condition confirmed before operating on the left side.

Zehender [5] was among the first to describe atresia of the puncta accurately. His patient was a healthy 12 year old girl who had suffered from lacrimation of the right eye from birth. Both puncta on the right and the one on the left lower lid were absent. The papillae were slightly marked, but there was a darker depression in the center which was covered with epithelium. The dark spot was punctured with a discission needle, and it was found to enter the canaliculus. Solutions injected into the canaliculus passed through the sac into the nose, and the lacrimation was relieved. In 1883, Zehender [6] described a similar condition in a 5 year old girl which yielded to the same treatment.

In 1877, Emmert [7] described the case of a youth, 18 years of age, in which all four puncta and canaliculi were absent. He made an artificial opening into the sac at the point of entrance of the inferior canaliculus below the anterior portion of the caruncle. By repeated probings he was able to keep the artificial opening patulous over a period of four months, during which time lacrimation was diminished. He did not report the final results.

Magnus,[8] in 1875, described the case of a healthy youth of 17 years with epiphora of both eyes, in whom both lower puncta were missing. The upper puncta were well developed. He described a second case [9] a few years later in a woman aged 21, in whom both lower puncta were absent. There was a dacryocystitis on the right, which was treated by slitting the upper canaliculus.

In 1884, Burnett [1] reported the case of a boy of 10 with a left eye that exhibited constant epiphora. The puncta of the left side were absent, the papillae being manifest only by a very slight elevation on the inner margins of the lid. The lower right punctum was absent;

4. Blanchet: Bull. Acad. roy. de méd. de Belgique **12**:621, 1846-1847.
5. Zehender: Klin. Monatsbl. f. Augenh. **5**:131, 1867.
6. Zehender: Klin. Monatsbl. f. Augenh. **21**:520, 1883.
7. Emmert: Arch. Ophth. **6**:525, 1877.
8. Magnus: Klin. Monatsbl. f. Augenh. **13**:199, 1875.
9. Magnus: Centralbl. f. prakt. Augenh. **4**:119, 1880.

the upper right punctum was present, though quite small. Burnett made an opening 2 mm. medial to the elevation and found a canaliculus without difficulty. He opened the canaliculus for about 2 mm., and as this remained patulous and no further epiphora occurred, further treatment was unnecessary.

Von Reuss,[10] in 1886, described the case of a boy of 12, a surviving twin who had suffered for many years from lacrimation. The papillae were present, but all four puncta were lacking. There was a tiny depression at the top of the prominence on the left lower lid; this was lacking on the upper lids. No treatment was given.

Fieuzal,[11] in 1887, described a case in which careful search failed to find any puncta. Incision was made at the place corresponding to the superior punctum and canaliculus after which, with a Stilling knife, an opening was made into the sac, and a permanent catheter was inserted. The patient was discharged three weeks later, and no result is reported.

DeWecker and Landolt stated in their textbook [12] that complete absence of the lacrimal puncta usually coincides with absence of the lacrimal glands. This imperforation is present sometimes in the superior puncta only, sometimes in both puncta of the same side. They cited the case of a man, aged 21, with an absence of all four puncta.

Cabannes [13] described the case of a man, 20 years of age, who had suffered all his life from lacrimation. Close examination failed to reveal papillae or puncta in the lower lids. About 2 mm. from the internal commissure was a cleft 1 mm. long over the course of the interior canaliculus. A no. 2 sound passed through this cleft into the sac. The upper puncta and canaliculi were normal.

Fox [14] reported the absence of the lower puncta in a patient who had not suffered from epiphora until he began to lead an outdoor life. An incision was made where the puncta should have been, and narrowed canaliculi were found. Small probes were passed with difficulty. By gradual dilatation, a full-sized probe was passed, and a no. 2 silver cannula was inserted. These were left in place, and the tears were carried away as rapidly as they were secreted.

Ferron,[15] in 1892, reported the case of a patient with chronic conjunctivitis in which no trace of puncta could be found. He said that he had often seen excessive atresia of the puncta in cases of chronic conjunctivitis but never a complete obliteration. He thought it probable

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10. von Reuss: Wien. med. Presse **27**:205, 1886.

11. Fieuzal: Bull. clin. nat. opht. **5**:195, 1887.

12. de Wecker, L., and Landolt, E.: Traité complet d'ophtalmologie, Paris, Vve. A. Delahaye et Cie, 1889, vol. 4, p. 1057.

13. Cabannes: Arch. d'opht. **16**:423, 1896.

14. Fox: Ophth. Rec. **9**:403, 1900.

15. Ferron: Bull. Soc. d'anat. et physiol. de Bordeaux **13**:87, 1892.

that the canaliculi were also absent, but had no chance of making sure as the patient refused all treatment.

Lafite [16] described the case of a girl with complete absence of the puncta on the left side and the lower punctum on the right. The patient's mother and maternal grandfather had always suffered from lacrimation, and a brother had been operated on for cyst of the lacrimal sac which had developed in connection with imperforation of the canaliculus on that side.

Lebegue,[17] in his thesis, reviewed the literature up to date and described five cases that had come under his observation and that of his confrères. These consisted of: 1. An infant with both lower puncta absent. 2. A girl of 9 with all four puncta absent, who was operated on without success. 3. A girl of 18 in whom the left inferior punctum was absent, with a slight depression where it should be. A probe was inserted with difficulty, but the patient failed to return to have a style put in place, and the tissues cicatrized. Later the accessory lacrimal gland was removed, which stopped the epiphora. 4. A girl of 18 with congenital imperforation of the lower left and upper right puncta, in connection with slight epicanthus. This patient had one blue and one brown eye. The puncta were opened, and internal canthoplasty was performed with amelioration of symptoms. 5. A boy of 19 with congenital absence of puncta on the left, those on the right being normal.

Kraupa,[18] in 1910, reviewed the literature and added the following case: In a boy, aged 14, the right upper punctum, as well as the left upper and lower puncta, was absent. Incision in the location of the left lower canaliculus proved its absence. Kraupa made an incision into the sac through the conjunctiva at the internal canthus, and left a permanent catheter in for several days, then removed it and replaced it daily for three months. Several months later the opening had closed. A second operation was done by opening the sac anteriorly and passing a probe into the sac. The conjunctiva was incised a little anterior to the crescentic fold, and a conjunctival flap was made. An incision was made between the caruncle and the crescentic fold to the sound in the sac. The conjunctival flap was drawn through this wound and fastened to the posterior margin of the lateral wall of the sac by means of a suture attached to the apex of the flap. The suture was brought out through the incision in the skin over the sac. According to Kraupa, this was a rather difficult procedure. A Hess permanent catheter was introduced and left in place for nine days, after which the catheter and sutures were removed. The late results were excellent, as there

16. Lafite: Bull. Soc. d'anat. et physiol. de Bordeaux **16**:34, 1895.

17. Lebegue: Anomalies et maladies congénitales des voies lacrymales, Thesis, Paris, 1910.

18. Kraupa: Klin. Monatsbl. f. Augenh. **48**:445, 1910.

was a funnel-shaped depression between the caruncle and the semilunar fold, which remained patulous. When examined a year later, the patient said that he had been free from lacrimation for the whole period.

Gradle [19] described a case of congenital atresia of the puncta on the left side in a woman aged 21. The papillae were present as small dark points, the puncta being covered with a thin layer of epithelium. This was perforated and a dilator inserted. Fluid was injected into the nose through both puncta, and there was no return of lacrimation after four months.

Blackmar [20] reported the case of a boy aged 11 years with all four puncta absent, and with probable absence of the salivary glands. He was unable to find the canaliculi by dissection, and considered the case to be one of congenital absence of the puncta and canaliculi associated with absence of the lacrimal glands.

COMMENT

The case here reported is unique in certain details. There was a complete absence of puncta and papillae of the left side, and a thorough search for the lower canaliculus revealed none. No search was made for the upper one, but it is fair to assume that none was present. I have found no record of the type of operation I have described, although the operation in Kraupa's case is somewhat similar. There is a history of epiphora in my patient after she applied herself to continuous use of her eyes, when it became very annoying. I doubted at first that the artificial opening would epithelialize and remain patent, and this opinion was shared by all ophthalmologists to whom I described the operation. Later observation has demonstrated that this operation has completely relieved the patient of epiphora. The technic is so simple and the results are so satisfactory that I wish to place the case on record so that other ophthalmologists confronted by a similar condition may have the benefit of my experience.

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19. Gradle, H. S.: Congenital Atresia of the Puncta Lacrimalia of One Side, Arch. Ophth. **50**:349 (July) 1921.

20. Blackmar, F. B.: Am. J. Ophth. **8**:139, 1925.

EXOPHTHALMOS IN INFANTILE SCURVY*

JOHN HUGHES DUNNINGTON, M.D.
NEW YORK

While it is difficult to state with any certainty the earliest description of scurvy, "the reference of Hippocrates to a large number of men in the army who suffered from pains in the legs, and gangrene of the gums, which was accompanied by loss of teeth, seems sufficiently definite to be identified as this disease" (Hess [1]). Its occurrence in infants was first recorded by Glisson in 1668, but not until 1862 was there any mention of exophthalmos as a complication, when Moeller [2] reported its presence in a case of "acute rickets" which, in view of subsequent knowledge, was undoubtedly one of scurvy. Magnus,[3] in 1878, was the next to record exophthalmos in infantile scurvy; however, little was known of the disease until 1883 when Barlow [4] published his classic paper giving anatomic proof that this disorder of infants showed the pathologic changes characteristic of adult scurvy. This article aroused great interest, particularly in England and in America, and many cases of scurvy were reported, but few contained any reference to exophthalmos.

In 1898 the comprehensive investigation of the American Pediatric Society [5] appeared, based on 379 cases of infantile scurvy, with the statement that swelling and protrusion of the eye were noted in forty instances. Still [6] reported the orbit involved six times in sixty-four cases, and Jacobi [7] four times in forty cases. Series such as these have led to the general statement in textbooks of pediatrics that exophthalmos occurs in approximately 10 per cent of cases of infantile scurvy. I believe that this is an erroneous impression, for in most of these cases the complication was merely mentioned without any description of the severity

* Submitted for publication, May 26, 1931.

* Read before the American Ophthalmological Society, Asheville, N. C., June 1, 1931.

* From the Department of Ophthalmology, Columbia University College of Physicians and Surgeons.

1. Hess: Scurvy Past and Present, Philadelphia, J. B. Lippincott Company, 1920.
2. Moeller: Königsb. med. Jahrb. **3**:135, 1862.
3. Magnus: Deutsche med. Wchnschr. **4**:365, 1878.
4. Barlow: Tr. Roy. Med. & Clin. Soc., London **66**:159, 1883.
5. Arch. Pediat. **15**:481, 1898.
6. Still: Common Disorders and Diseases of Childhood, ed. 5, London, H. Milford, 1927, p. 140.
7. Jacobi: Diseases of Children, New York, D. Appleton and Company, 1909, p. 219.

of the involvement. Therefore, it is probable that the exophthalmos, if genuine, was not marked. Detailed case reports are rare, for DeBuys[8] in reporting a case of his own in 1912 could find only twelve instances in which sufficient data had been given to enable him to include them in his tabulation. This comprehensive article includes a summary of the cases reported up to that time and is the most illuminating one on this subject. A careful review of the literature reveals nine more (Holmes Spicer,[9] Dewey,[10] Blake,[11] Jost,[12] Place,[13] Tallei [14] and Van Duyse [15]); so it can be said that to date only twenty-two detailed case reports of exophthalmos in scurvy are on record. Of course, there are many other references to its occurrence, but they do not contain sufficient data to enable one to draw any conclusions from a study of them. This paucity of detailed ocular reports is my chief reason for presenting this subject.

Infantile scurvy was once thought to be a disease of the bone marrow, but the investigations of Aschoff and Koch,[16] Hess and others have shown it to result from a defect in the cement substance of the endothelial cells of the blood vessels produced by a lack of vitamin C in the food. This increase in permeability of the blood vessel walls is an important factor in the production of hemorrhage, which is responsible for so many of the symptoms of this disease, among them being exophthalmos.

The direction of the proptosis is the most constant feature of the exophthalmos, the eye being pushed forward, downward and outward, and this is readily understood when one considers the actual lesion. While several authors have contended that hemorrhage into the loose areolar tissues of the orbit may account for some of the protrusions of the eyeball, Barlow [17] in 1889 expressed the belief that extravasation of blood between the orbital plate of the frontal bone and the subjacent periosteum produces it. The truth of Barlow's contention was substantiated in 1896 by Meyer,[18] who, in a case of extreme exophthalmos, made an incision along the upper orbital margin. With a probe he felt the orbital roof free from periosteum, and later corroborated this at autopsy. Snow [19] also proved by autopsy that a huge hematoma of sub-

8. DeBuys, L. R.: Exophthalmos in Scurvy, J. A. M. A. **59**:2040 (Dec. 7) 1912.
9. Holmes Spicer, W. T.: Tr. Ophth. Soc. U. Kingdom **12**:33, 1892.
10. Dewey: Ann. Ophth. **20**:307, 1911.
11. Blake: Am. J. Ophth. **4**:736, 1921.
12. Jost: Bull. Soc. d'opht. de Paris, 1926, p. 714.
13. Place: Am. J. Ophth. **8**:955, 1925.
14. Tallei: Boll. d'ocul. **4**:247, 1925.
15. Van Duyse: Bull. Soc. belge d'opht., 1928, no. 57, p. 51.
16. Aschoff and Koch, quoted by Ratnoff: Arch. Pediat. **42**:183, 1925.
17. Barlow, in Keating: Cyclopedia of the Diseases of Children, Philadelphia, J. B. Lippincott Company, 1889, vol. 2, p. 265.
18. Meyer: Berl. klin. Wchnschr., 1896, p. 85.
19. Snow: Arch. Pediat. **22**:576, 1905.

periosteal origin was responsible for the exophthalmos in his case. So far as is known, calcification of these subperiosteal orbital hemorrhages has never been demonstrated by x-rays. While the extreme nature of these cases may cause some to doubt that subperiosteal hemorrhages are responsible for all proptoses, is it not true that scorbutic hemorrhages usually occur where there is either physiologic growth or pathologic irritation? Certainly, neither of these conditions prevails in the soft tissues of the orbit. The subperiosteal hemorrhages occur most frequently along the orbital plate of the frontal bone, although Place, by mistaking his case for one of orbital cellulitis, explored the orbit to find the orbital plate of the ethmoid denuded of its periosteum over practically its entire area and a large cavity existing between the bone and the periosteum. No case appears in the literature in which only the floor of the orbit has been involved, so in all of the cases the displacement has been forward, downward and outward.

The hemorrhagic origin of the exophthalmos easily explains its sudden appearance and also accounts for its proneness to recur unless treatment is instituted promptly. Hart and Lessing,[20] in experimental scurvy in monkeys, noted hemorrhages into the orbit severe enough to cause exophthalmos, but for the purposes of this study the most interesting experiment is that of Harden and Zilva.[21] These investigators produced scurvy in a monkey, and noted exophthalmos eight days after the appearance of the initial scorbutic lesion. The exophthalmos disappeared in two or three days, leaving the upper lid and orbit blood-stained, only to reappear in about ten days more pronounced than ever. This proptosis also disappeared in two days, although no change had been made in the diet during all this time. The general condition of the monkey became so grave that antiscorbutic treatment was then instituted, with complete recovery in about a week. A similar type of history was obtained by DeBuys in his case in which two attacks of exophthalmos cleared without antiscorbutic treatment, only to recur in a much severer form. Zilva and Still,[22] after feeding a monkey for two months on a scorbutic diet, noted the appearance of exophthalmos and hemorrhage into the lids. After curing the condition in three days by adding decitrated lemon juice to the diet, they caused it to reappear in a month by omitting the lemon juice, then promptly cured it again. While spontaneous absorption of the hemorrhages does take place, relapses are prone to occur unless the antiscorbutic diet is continued.

It has been contended that absorption of these orbital hemorrhages is very slow. Holmes Spicer, in 1892, reported a case in which after

20. Hart and Lessing, quoted by Hess (footnote 1, p. 127).

21. Harden and Zilva: J. Path. & Bact. **22**:249, 1919.

22. Zilva and Still: Lancet **1**:1008, 1920.

seven months one eye was still abnormally prominent. However, judging from an analysis of the cases reported since 1912, the duration of the exophthalmos is extremely short after antiscorbutic treatment is once started. The longest length of time required for complete recovery in these cases was one month (Van Duyse) and the shortest, four days (Blake). Earlier diagnosis and more energetic treatment probably account for the more rapid recoveries.

The varying severity of the exophthalmos in these cases is striking. Every conceivable degree of bulging has been noted from the slightest swelling to the most marked exophthalmos. Those recorded as slight protrusions are open to criticism, for it is evident that such a diagnosis is difficult either to make or to dispute. Inasmuch as edema of the eyelids is a frequent finding in the subacute or latent type of infantile scurvy, is it not possible that some of the so-called mild proptoses were of this nature? The exophthalmos may be sufficiently pronounced to endanger the cornea. A large corneal erosion complicating an extreme exophthalmos led Jost to puncture the orbit and do a total tarsorrhaphy rather than wait for the effects of antiscorbutic treatment; the result justified the procedure, while conservative measures in a similar case of Tallei's resulted in a dense corneal leukoma. Van der Straeten[23] reported corneal ulceration with perforation as a complication in an extreme case. If the hemorrhage is confined entirely within the orbit, the exophthalmos is not accompanied by any lid involvement (DeBuys, Blake). In most of the cases, however, the blood oozes in front of the septum orbitale, producing ecchymosis or suggillation of the lids, particularly the upper one. The swelling of the lid may become so tremendous as to mask the actual protrusion of the eye (fig. 1). The exophthalmos usually precedes the lid involvement, or else the two occur at the same time.

The exophthalmos is nonpulsatile and irreducible, and any attempt at reduction causes great pain. Such manipulation may precipitate a hemorrhage into the lid or cause an increase in the proptosis. The proptosis may involve one eye or both; usually if it is bilateral, one eye is affected before the other. In confirmation of Still's statement that the left eye is involved more frequently than the right, my analysis of seventeen unilateral cases showed the left eye to be affected eleven times while the right eye was involved in only six instances. Most writers have stated that the motility of the eye is not impaired, but Jost found the eye to be immobile in his extreme case. It seems as if the impairment of motility should be in direct proportion to the severity of the exophthalmos. While its onset after a prolonged spell of crying was noted by Holmes Spicer, DeBuys and others, I agree with Place that the

23. Van der Straeten: Bull. Soc. belge d'opht., 1928, no. 57, p. 52.

crying was probably the effect rather than the cause. The occurrence of impaired motility is usually a late manifestation of scurvy; however, Barlow and Schlesinger [24] have recorded its presence as the initial sign of this disease. Diagnostic methods have improved so markedly during the twenty-five years since this last report that it is highly improbable that any one will ever be forced to make a diagnosis of scurvy on the presence of exophthalmos alone. It is important, however, to bear in mind that this complication may arise before the classic symptoms are present.

AGE INCIDENCE

While scurvy occurs at all ages, the complication of exophthalmos usually arises in patients less than a year old. The average age of the patients in the twenty-two authentic cases is 10½ months, the youngest being 7 months and the oldest 2 years. Reyher [25] referred to having seen it in a 7 year old scorbutic boy. Krükow,[26] in his studies on scurvy in Russia, reported a case of recurrent orbital hemorrhage in an adult; this is the only reference in the literature to the occurrence of this complication in the scurvy of adults.

The rapidity of growth of the orbits within the first few months doubtless accounts for the rather frequent occurrence of exophthalmos in infantile scurvy, for, as previously stated, subperiosteal hemorrhage is particularly prone to occur at the site of rapid physiologic growth. With this in view, a comparison of the dimensions of the orbit at the time of birth and at the end of the first eighteen months is very instructive. Whitnall [27] gave the following figures:

	Margin		Depth			
Age	Height, Mm.	Width, Mm.	Roof, Mm.	Floor, Mm.	Lateral Wall, Mm.	Medial Wall, Mm.
Birth.....	18	21	32	25	27.5	22.5
18 months.....	30	32	46	40	38	36.5
22 years.....	36	37	48	45.5	43	39
Adult male.....	33	39	49.5	52.5	47	39
Adult female...	34	36	45	43.5	42	34

A study of these figures reveals a very rapid increase in the size of the orbit during the first year and a half; hence it is a particularly favorable site for the occurrence of a subperiosteal hemorrhage in this disease.

24. Schlesinger: München. med. Wchnschr., 1905, no. 43, p. 2073.

25. Reyher, in Kraus and Brugsch: Spezielle Pathologie und Therapie, Berlin, Urban & Schwarzenberg, vol. 9, pt. 1, p. 672.

26. Krükow, in Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, vol. 14, pt. 7, p. 204.

27. Whitnall: Anatomy of the Human Orbit, New York, Oxford University Press, 1921, p. 99.

Although orbital hemorrhage is the striking ocular lesion of infantile scurvy, hemorrhages into the lid, conjunctiva, anterior chamber and retina have been described. Stephenson [28] recorded a case of hematoma of the lower lid without orbital involvement. Barlow, Steindorff [29] and others reported cases of subconjunctival hemorrhage, Kaltz [30] one of hemorrhage into the anterior chamber and Hirschberg [31] one of retinal hemorrhages as an accompaniment of an orbital hemorrhage.

REPORT OF A CASE

History.—A. G., a white girl, aged 7 months, was referred to me by Dr. Martin T. Quinn because of swelling of the left eye of one week's duration. The birth was normal, and the child had been perfectly healthy until two weeks prior to the onset of the present illness, when the mother noticed that the patient was fretful and had a poor appetite. She was breast-fed for one month and then fed on a rather large formula containing 39 ounces (1105.6 Gm.) of whole milk, 11

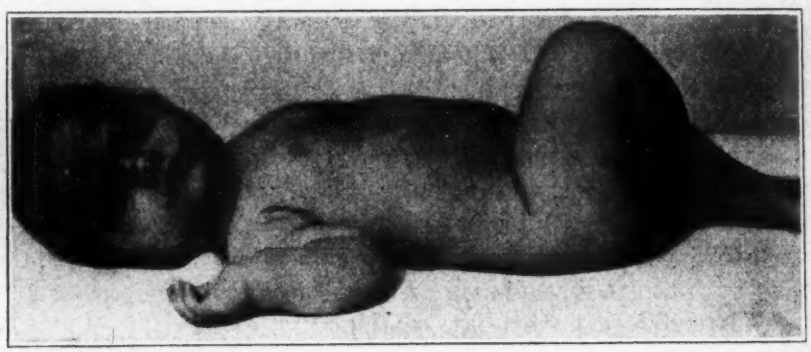

Fig. 1.—Marked hematoma of the lid masking the exophthalmos. Characteristic flexion of the right leg.

ounces (311.8 Gm.) of water and 2 ounces (56.7 Gm.) of dextrose maltose. She had taken a teaspoonful of cod liver oil a day since 1 month of age, and had also had 10 drops of orange juice "between feedings." Eleven days prior to examination she had a slight "head cold" which after four days was complicated by sudden swelling of the left upper eyelid and bulging of the left eye. This largely disappeared in three days, but recurred four days later. During this time and for one week prior to the illness the child cried almost constantly, particularly when her legs were handled. Her rectal temperature had never been higher than 100 F. Her appetite was poor; she was constipated and appeared to be in pain.

Examination.—The child was well developed and not anemic. The right eye was normal. The left upper lid was markedly swollen, ecchymotic (fig. 1) and extremely tender to the touch. The eye was proptosed, being pushed downward,

28. Stephenson: Ophthalmoscope **13**:134, 1915.

29. Steindorff: Ztschr. f. Augenh. **25**:180, 1911.

30. Kaltz, quoted by Steindorff (footnote 29).

31. Hirschberg: Centralbl. f. prakt. Augenh., 1903, p. 206.

forward and outward. The exophthalmometer reading was not satisfactory, but I estimated the amount of exophthalmos as 2 mm. The movements of the eye appeared limited in upward gaze, but this, too, was difficult to ascertain with any degree of certainty. The conjunctiva, cornea, iris, media and fundus were normal. The child lay with her legs flexed, and extension caused great pain. The skin was clear except for a few small ecchymotic spots on the buttocks. The results of examination of the ear, nose and throat were negative, as was a roentgenogram of the nasal accessory sinuses. The upper gums were spongy and bleeding, and the lower ones were slightly ecchymotic (fig. 2). The chest was normal except for a scorbutic rosary. The heart was rapid, but the sounds were of good quality. The spleen and liver were slightly enlarged. Both thighs were swollen and markedly tender. The results of the examination of the blood were: hemoglobin, 72 per cent; red blood corpuscles, 4,408,000; white blood corpuscles, 11,300; polymorphonuclears, 68 per cent; lymphocytes, 32 per cent; coagulation time, 2½ minutes, and bleeding time, 1 minute. The urine contained albumin and numerous red blood cells.

Roentgen Findings.—Complete roentgenograms were taken by Dr. John P. Caffey whose findings follow. On Nov. 3, 1930, the long bones were examined.

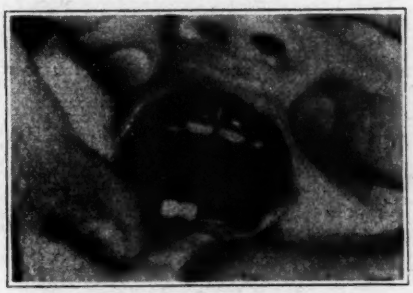

Fig. 2.—Typical scorbutic changes of the gums.

Upper Extremities: The bones appeared normal throughout. There were no diagnostic scorbutic changes present. The ground glass appearance of the diaphyses was moderately suggestive of early scurvy.

Lower Extremities: The provisional zones of calcification of the distal ends of both femora were definitely increased in both width and density. Similar changes were also present at the distal ends of both tibiae and fibulae. There were no well developed zones of submetaphyseal rarefaction in any of the long bones. There was no cortical atrophy. The ossification centers in the epiphyses did not show "edging" with central rarefaction as is seen in typical well developed scurvy. Spurs were visible at the distal ends of both femora.

Thorax: There was a definite increase in the width and density of the primary zones of calcification at the costochondral junctions, with an associated increase in the density of the shaft of the ribs at this point. The changes suggested a moderate scorbutic rosary.

Skull: The left orbit was obscured by a moderately hazy increase in density which is consistent with orbital hemorrhage in scurvy. This change in the orbit, however, could not be differentiated roentgenologically from other lesions, either inflammatory or neoplastic, which might cause a similar increase in the soft tissue density of the left orbit. The right orbit appeared normal.

Impression: The changes in the primary zones of calcification in the femora, tibiae and fibulae and the spurs at the edges of the distal metaphyses of the femora were practically diagnostic of scurvy. One could prognosticate roentgenologically that the administration of orange juice would probably cause calcification of subperiosteal hemorrhage along the distal ends of both femora because of the "spurs" that were present in these locations. It was noteworthy that the changes in the ossification centers which are usually so prominent in scurvy had not developed yet. This, with the fact that there were practically no roentgenologic lesions in the

Fig. 3.—Roentgenograms of the lower extremities taken four weeks after the onset of the condition, showing calcification of the subperiosteal hemorrhages at the distal end of the femora.

bones of the upper extremities, indicated that, while the changes seen were consistent with scurvy, they represented the early first stage of that disease.

Report on follow-up films made on Jan. 31, 1931 follows. Upper Extremities: No abnormalities were seen.

Lower Extremities: The medial border of the distal end of the right femur showed an irregular outline of increase in the width of the bone. The distal end of this bone also showed a general increase in density with a loss of definition of the trabecular pattern. The ossification centers of the distal epiphyses of the femur showed a beginning central rarefied nucleus.

Impression: These changes were compatible with and, in fact, practically diagnostic of the fourth, or healing, stage of scurvy. A review of the evidence in previous films of active early scurvy taken in comparison with the changes present at this time indicated that the roentgenologic lesions seen in these bones at the two different examinations made the diagnosis of scurvy a certain one.

A stereoroentgenogram of the skull on April 6, 1931, revealed no evidence of calcifying hemorrhage in the left orbit. The haziness that was present previously, on the left, had cleared.

With the diagnosis of scurvy established, the child was placed on 3 ounces (88.76 Gm.) of orange juice daily, which was followed by a rapid disappearance of the swelling of the upper lid, of the proptosis and of the tenderness along the femora. The improvement was so rapid that in four days the eye appeared practicaliy normal, and in a week the child left the hospital in excellent condition. The therapeutic test along with the other positive findings definitely establishes this case as one of infantile scurvy.

SUMMARY

Infantile scurvy is not an infrequent cause of exophthalmos in children less than 2 years old. The direction of the proptosed eye is uniformly forward, downward and outward. The sudden onset of the condition is characteristic, and its hemorrhagic origin is undisputed. The diagnosis is easily made, and rapid and complete recovery follows antiscorbutic treatment.

CYTOLOGIC STUDIES ON THE RETINA

I. NORMAL COEXISTENCE OF OLIGODENDROGLIA AND MYELINATED NERVE FIBERS *

MILTON L. BERLINER, M.D.
NEW YORK

In a recent paper entitled "Extensive Medullated Nerve Fibers Associated with Choroiditis," [1] I suggested the idea that the sporadic and anomalous appearance of medullated nerve fibers in the human retina might be due to the abnormal presence of one of the types of glial cells, namely, the oligodendroglia. This view was advanced because of the increasing tendency to regard the oligodendroglia as the element having to do with the secretion of myelin.

In the optic nerve, as in the central nervous system, three types of neuroglia occur, namely, the astrocytes, the microglia and the oligodendroglia. The astroglia, with its two types, protoplasmic and fibrous, are found throughout the nervous system. This type of neuroglia is characterized by the fact that the cells have some prolongations, the ends of which are applied against the walls of the small vessels. The microglia, in all probability mesodermic in origin, are phagocytic and correspond to the histiocytes of the connective tissue system. The oligodendroglia are seen in rows and are especially conspicuous in the white matter where their prolongations envelop the nerve fibers. In the retina of man, where there is normally no myelinization there are no oligodendroglia formed; the only glial cells that appear are the astrocytes and the microglia. The intervention of the latter in several pathologic processes of the human eye has been described by Lopez Enriquez.[2]

In order to determine whether the presence of medullated nerve fibers is constantly correlated with the existence of oligodendroglia, it seemed advisable to study the retina of an animal in which myelinated fibers are normally found. The retina of the rabbit fulfils this condition. Furthermore, since myelinated nerve fibers occur in a more or less restricted area in the retina of this animal, it allows room for comparison with other areas in which there is no myelinization.

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* From the Department of Anatomy, Cornell University Medical College.

1. Berliner, M. L.: Extensive Medullated Nerve Fibers Associated with Choroiditis: Report of a Case, Arch. Ophth., to be published.

2. Lopez Enriquez, M.: Arch. de oftal. hispano-am. **27**:322, 1927.

MACROSCOPIC DESCRIPTION OF THE NORMAL FUNDUS OF THE RABBIT

The nerve head is situated considerably below the posterior pole of the eyeball. It is completely excavated. The sides of the excavation slope sharply downward into a pit without any undermining. The long axis of the nerve head lies in the horizontal plane (fig. 1). There being no lamina cribosa present, the opaque medullated nerve fibers are seen

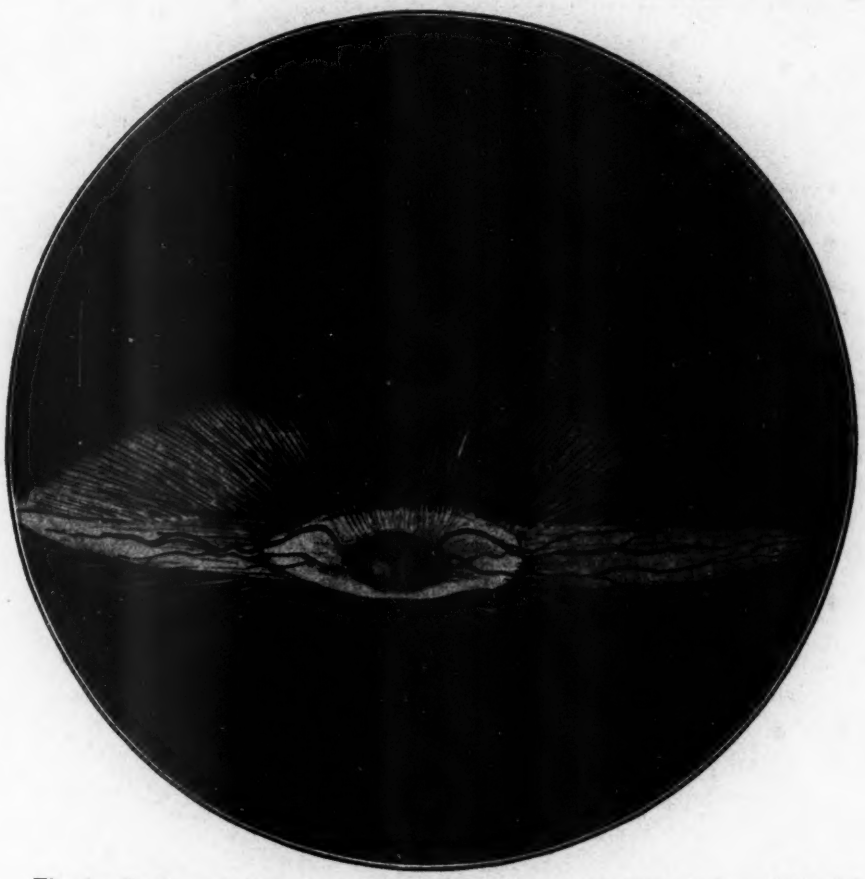

Fig. 1.—Fundus of the eye of the rabbit (after preservation in formaldehyde) showing physiologic excavation and areas of myelinated nerve fibers.

emerging en masse from the pit of the nerve head. As they extend over the margin of the crater of the excavation, they form a thick white mound, particularly on the lateral sides. Above, the width of the mound is somewhat narrower, while below it is hardly present. Laterally, the opaque nerve fibers radiate from the distal edges of the mound in compact formation and extend outward for a considerable distance to the

periphery of the retina. In this region they tend to thin out and become feathery. Above, the opaque nerve fibers leave the mound abruptly in thin, straight bundles, separate from one another, and extend for only a very short distance. Below, a scattered few opaque fibers are barely seen leaving the mound, where they abruptly lose their medullations. The appearance of the nerve head with its lateral expansions of opaque nerve fibers resembles that of a two-bladed propeller.

MICROSCOPIC APPEARANCE OF NERVE HEAD AND RETINA OF THE RABBIT

In sagittal sections of the posterior segment of the globe fixed in Heidenhain's trichloracetic acid-sublimate-formaldehyde mixture and stained with hematoxylin-eosin, the distribution of the glial nuclei can be readily seen in more or less columnar formation. As there is an absence of lamina cribrosa, these cells meet no resistance and continue directly from the optic nerve and extend into the retina, particularly in the horizontal meridian. These rows of oligodendroglia in the retina are especially seen where the myelinization is heaviest, i. e., near the disk laterally (fig. 2 *B*). As the myelinated fibers thin out, the nuclei of the oligodendroglia become scarce; and in those areas in which the nerve fibers are nonmedullated, a complete absence of the characteristic rows of oligodendroglia is found. Also, the nerve fiber layer itself becomes much thinner, and there are only a few scattered nuclei visible. Above and below, where the medullated nerve fibers are less compact, and where they extend for only a very short distance, there is a marked paucity of cells.

In cross-sections of the lateral expansions of the nerve head, the arrangement of the nerve fibers into bundles is clearly seen (fig. 2 *A*). The bundles are separated from each other by the compressed cells of Mueller. Only a few oval nuclei are seen between the bundles. Some may belong to the cells of Mueller, while others are nuclei of astrocytes. Within the bundles, the round nuclei of the oligodendrocytes appear irregularly scattered. Since the section is transverse, their arrangement in series is not obvious, but is indicated by a slight overlapping of some of the nuclei.

A comparison between the nerve bundles under consideration and the corresponding bundles in the optic nerve shows no essential difference. The nerve fibers are of variable thickness and have no definite arrangement in the bundles. The variability in thickness of the myelin sheath as it surrounds each fiber is best seen in cross-sections of the bundles of fibers in material prepared with the silver carbonate method (fig. 3).

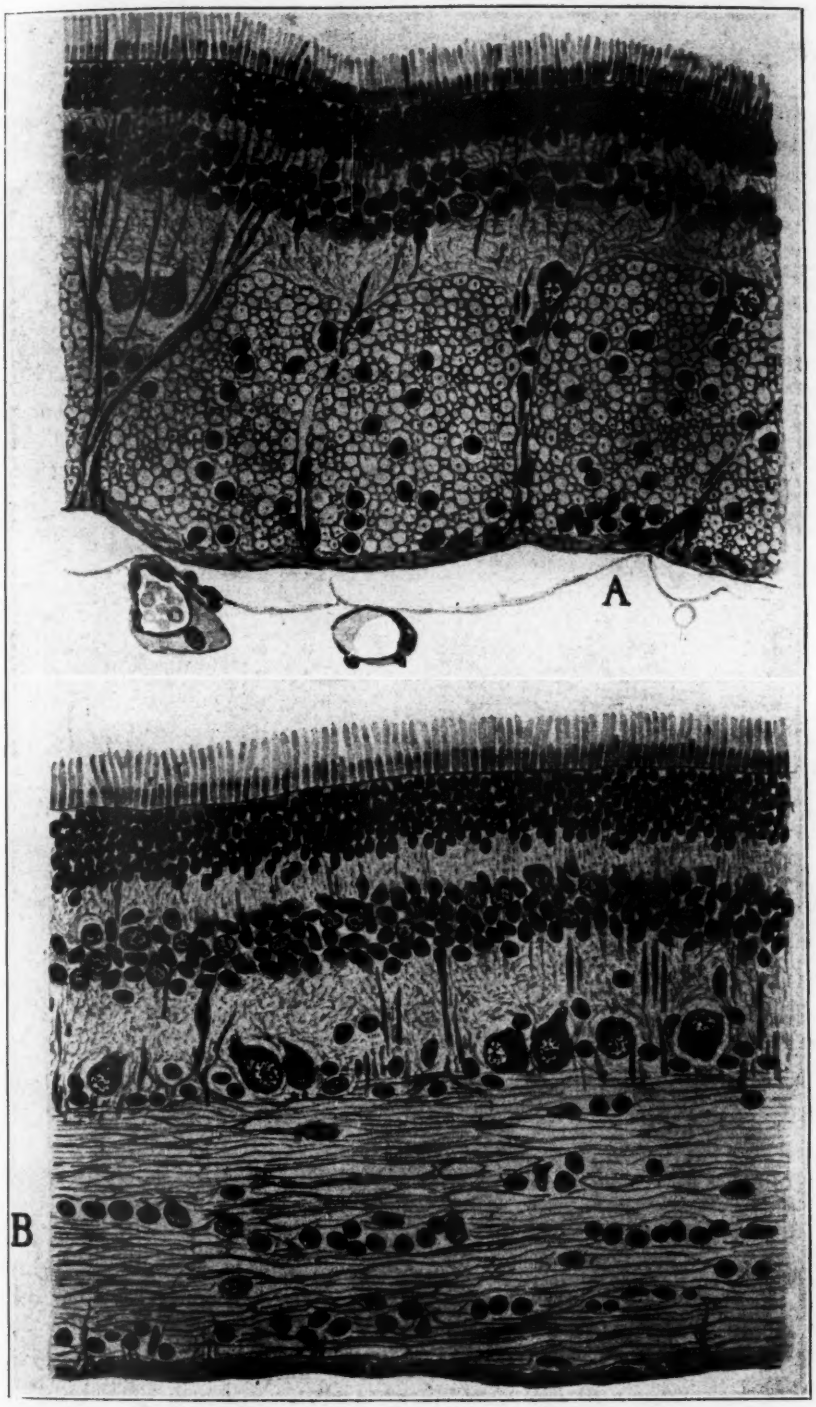

Fig. 2.—*A*, a portion of a sagittal section of the retina of the rabbit passing through one of the lateral expansions of the myelinated fibers. The myelinated fibers appear in cross-section and are grouped into large bundles. Note the numerous oligodendroglia nuclei, scattered in the bundles. *B*, a portion of the same retina showing myelinated fibers in longitudinal section. Notice the rows of oligodendroglia nuclei in the nerve fiber layer. Heidenhain's sublimate-trichloracetic fixation. Hematoxylin-eosin.

In order to prove that the rows of cells seen in the retina correspond to those in the optic nerve, which, as shown by Lopez Enriquez,[3] are formed chiefly by oligodendroglia, I have used a specific technic for staining the oligodendroglia.

TECHNIC FOLLOWED FOR THE DEMONSTRATION OF THE CHARACTERISTICS OF THE OLIGODENDROGLIA IN THE RETINA OF THE RABBIT

The method of Rio Hortega (silver carbonate) gives best results when applied to frozen sections. However, in view of the fact that it is extremely difficult to obtain frozen sections of the retina, I have sought to modify the method. Since the retina is so thin, it was found best to dissect it out in one piece, being careful

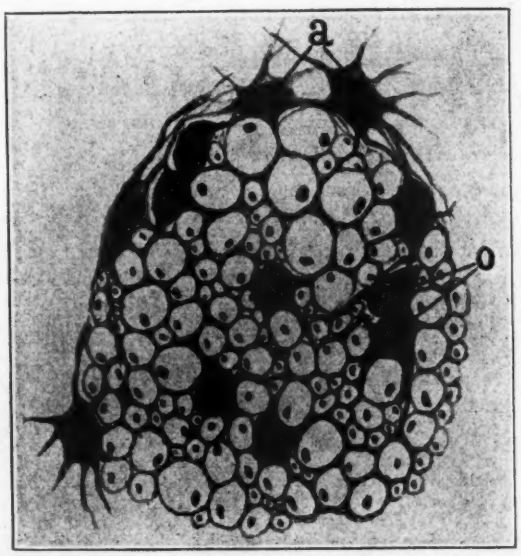

Fig. 3.—Myelinated fibers in cross-section showing axons and oligodendroglia cells (*o*), astrocytes (*a*). Rio Hortega's method (for oligodendroglia).

not to tear it from its attachment to the optic nerve. The formula for the staining of the oligodendroglia, as given by Rio Hortega,[4] was used.

1. The eyes of the rabbit were fixed in the following solution: a diluted solution of formaldehyde U. S. P. (1:10), 100 cc.; chloral hydrate, 2 Gm., and ammonium bromide, 1 Gm. The best results were obtained in the case of two eyes kept in this solution for ten days.

2. The eyes were then rinsed with water. The optic nerves, with the attached retinas, were dissected from the other coats of the eye and were washed in two or three changes of distilled water.

3. The retinas were immersed in distilled water to which two or three drops of ammonia had been added (from ten to fifteen minutes).

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3. Lopez Enriquez, M.: Bol. Soc. españ. de hist. nat. **26**:301, 1926.
4. del Rio Hortega, P.: Mem. r. Soc. españ. de hist. nat. **14**:5, 1926.

4. The retinas were immersed in a strong silver carbonate solution which was prepared as follows: 5 cc. of a 10 per cent solution of silver nitrate is mixed with 20 cc. of a 5 per cent solution of silver carbonate. The heavy precipitate is slowly dissolved through the addition of ammonia, care being taken to avoid an excess of ammonia. Since these prepared retinas are thicker than most frozen sections,

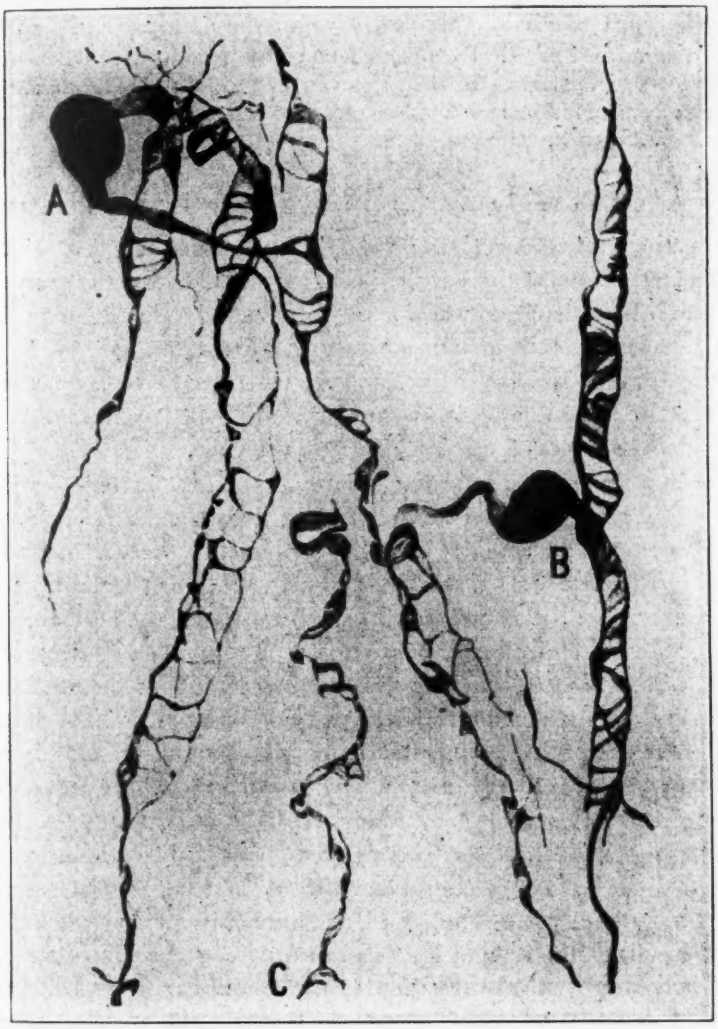

Fig. 4.—Oligodendrocytes of the medulla oblongata of the cat. *A*, cell with reticular prolongations wrapped around three myelinated nerve fibers; *B*, cell with lamellar prolongations surrounding a myelinated fiber; *C*, spiral-shaped prolongation of an oligodendrocyte. (After Rio Hortega.)

the silver solution was allowed to act for fifteen minutes at room temperature and then from fifteen to twenty minutes at 38 C.

5. Before the silver solution cooled off, the retinas were lifted from it with a glass rod and washed in 60 per cent alcohol for ten minutes.

6. When taken from the alcohol, the retinas were placed in a diluted solution of formaldehyde, U. S. P. (1:10) in which the reduction of the silver takes place in from five to ten minutes.

7. After thorough washing in several changes of distilled water, the tissues were toned with a 1:500 solution of gold chloride. They were then fixed in a 5 per cent solution of sodium thiosulphate. The retinas were again washed in distilled water, dehydrated in the alcohol, cleared with xylene and embedded in paraffin.

With this method excellent impregnations of the retinas were obtained, and in addition to the oligodendroglia, the astrocytes and cells of Mueller were stained. Furthermore, the method shows other structural details that cannot be seen in sections stained with hematoxylin-eosin.

THE OLIGODENDROGLIA IN THE RETINA OF THE RABBIT

In sections of the retinas, stained by the method just described, the characteristics and the peculiar behavior of the prolongations of the oligodendroglia are clearly seen. As shown by Rio Hortega for the central nervous system, the prolongations arise from the cell body and are of irregular thickness. In many places they appear with a lamellar or ribbon-like flatness and are wrapped around the myelinated fibers, as can be seen in figure 4, copied from this author.

The oligodendrogliocytes that surround the myelinated fibers in the retina of the rabbit behave in an identical manner. The prolongations of these cells are particularly conspicuous around the myelinated fibers as shown in figure 5. Since the staining of the cytoplasm is rather deep, the nuclei of the oligodendroglia cells are seen with difficulty. The appearance of the prolongations around the myelinated fibers varies somewhat according to whether the fibers are seen on the surface view or in optical section. In the latter case the contour of the fiber may be seen since the prolongations of the oligodendroglia are closely applied to the myelin sheaths of the fibers. The axons in the latter appear faintly stained. They are more easily identified as axons in cross-sections of the fibers (fig. 3 *A*). If the fiber is very thick, a delicate network with triangular nodes or a coarser network of lamellar prolongation will appear on surface view (fig. 5 *C*). Since the prolongations of the oligodendroglia extend for a long distance among the myelinated fibers, the cell bodies are not always seen in the vicinity of the prolongations, but can be located when the latter are traced. Some cells have elongated nuclei, especially when they occur among closely placed fibers (fig. 5 *E*).

The number of prolongations vary according to the size of the cell. Some, after leaving the cell body, run obliquely among the parallel nerve fibers and are wrapped around the myelin sheaths of fibers situated at some distance from the cells. In this way a relatively small number of oligodendroglia cells furnish wrappings for a large number of nerve fibers.

Where the myelinated nerve fibers are thin, the behavior of the prolongations is more clearly seen. They are closely wrapped around the sheaths of these fibers in a spiral fashion (fig. 5 *B, D, E*). In the retina of the rabbit the cells of the oligodendroglia form linear groups, and their prolongations are so closely packed that they produce a plexiform aspect. A group of these cells has been copied in figure 5 *A*.

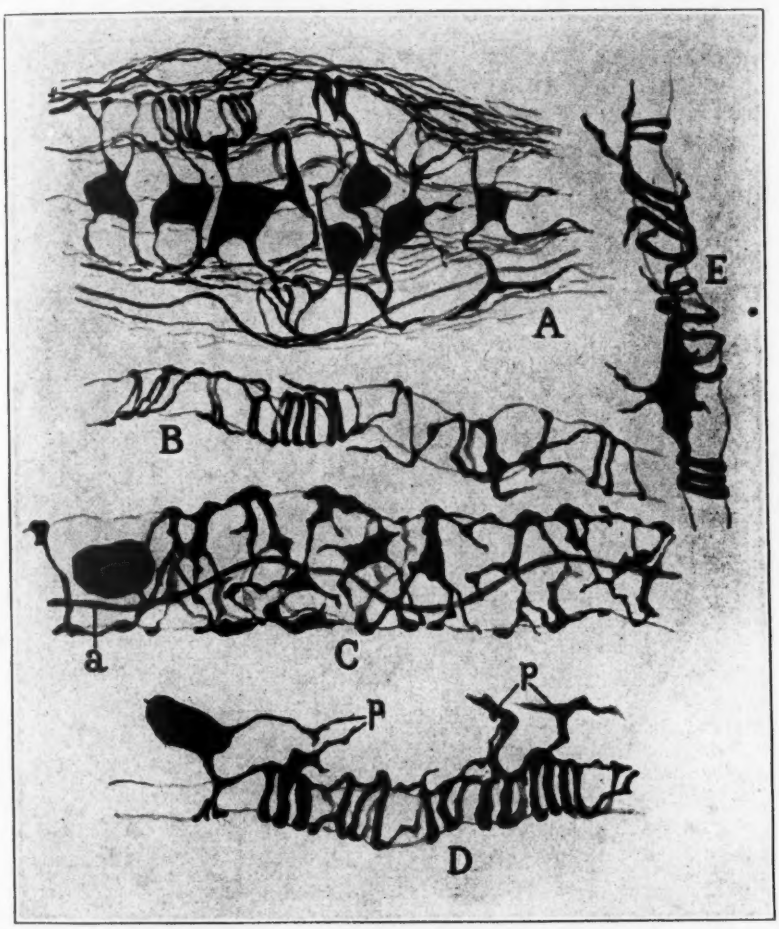

Fig. 5.—Diverse aspects of the oligodendroglia in the zone of myelinated fibers of the rabbit's retina. *A*, group of oligodendrocytes showing a few prolongations wrapped around medium-sized fibers; *B*, prolongations in the form of rings around a medium-sized fiber; *C*, oligodendrocyte with lamellar prolongations surrounding a thick myelinated fiber; *a*, axon of the fiber; *D*, oligodendrocyte with prolongations wrapped around a thick myelinated fiber; *p*, prolongations going to other fibers; *E*, oligodendrocyte with elongated nucleus, closely applied to a thin fiber. Rio Hortega's method (for oligodendroglia). Zeiss apochr. imm. 2 mm., ocul. 15.

Some of their prolongations are wrapped around the fibers in contact with the cells.

In addition to the processes of the oligodendroglia, numerous branches of the astrocytes are seen. The latter do not stain deeply. They are scattered irregularly among the myelinated fibers. In contrast to the oligodendroglia, they occur in portions of the retina that lack

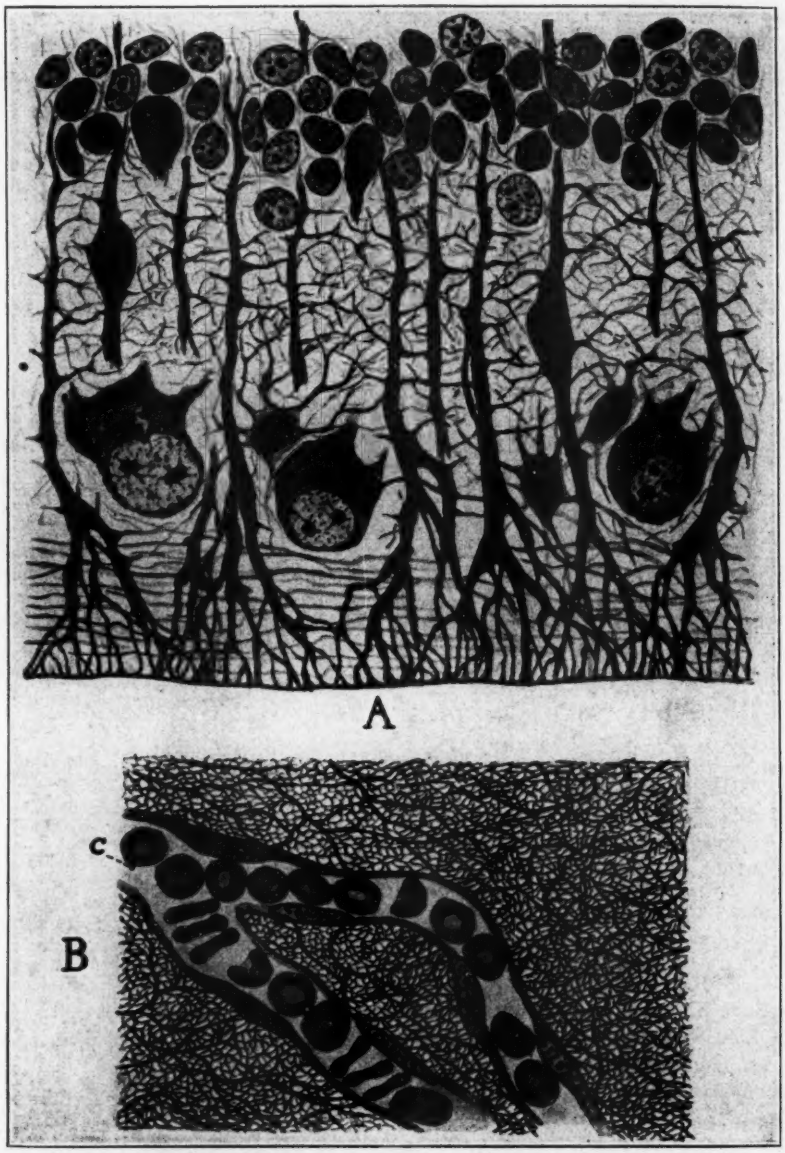

Fig. 6.—*A*, inner layers of the retina beyond the area occupied by the myelinated fibers. Three astrocytes are seen between the ganglion cells. Note the absence of oligodendroglia. *B*, surface view of the inner limiting membrane of the retina showing reticulated structure of the latter. *c*, blood capillary. Rio Hortega's method (for oligodendroglia). Zeiss apochr. imm. 2 mm, ocul. 15.

myelinated fibers (fig. 6 *A*). A certain number of deeply stained irregular nuclei probably belong to the microglia. Aside from a thin area around the nucleus, the cytoplasm of these cells does not stain. The stain used here is not specific for microglia. The portions of the retina lacking myelinated nerve fibers are very different in appearance when stained by the silver carbonate method. In the first place, as already stated, the nerve fiber layer is very thin. Secondly, few nuclei are seen in this layer. Those present belong to the astrocytes or to the microglia. In all sections the cells of Mueller, which are genetically related to the glia, appear well stained (fig. 6). The proximal portions of Mueller's fibers (near the limitans interna) are seen to consist of many fine branches. These form a meshwork of delicate trabeculae which fill the spaces between the cells in the upper layers of the retina. The occasional astrocyte found in the nerve fiber layer sends processes in all possible directions. Some of these reach the lamina interna and end there. When the limitans interna has been detached from the retina, owing to manipulations of the fixation, etc., and is seen on surface view, it appears as an extremely fine network of close meshes resembling a fenestrated membrane (fig. 6 *B*). The fibers that constitute the network stain deeply with silver carbonate. It is questionable, in fact, whether the limitans interna is a continuous structure. When seen in sections, it appears as a basement membrane. This may be the result of an optical illusion. The silver carbonate staining indicates that the membrane is formed by fine prolongations from the branches of the cells of Mueller and the astrocytes. A similar condition occurs during the development of the central nervous system. The external limiting membrane of the brain and cord is formed by an interlacing of the prolongations of the developing astroglia cells. A membrane of this type would not prevent the passage of fluid or toxins from the vitreous body. The limiting membrane of the central nervous system permits absorption of the cerebrospinal fluid. This comparison is justified since the retina is a modified portion of the brain.

From the preceding descriptions, it is clear that the cells situated between the myelinated fibers of the retina of the rabbit are identical with the oligodendroglia of the central nervous system. The identity is manifested in the similarity of the staining reactions, and the character of the prolongations, particularly the manner in which they envelop the myelin sheaths of the nerve fibers.

COMMENT

The evidence presented in this paper clearly shows that my former assumption that the presence of myelinated fibers in the retina is correlated with the occurrence of oligodendroglia is essentially correct. In

the central nervous system where myelinated fibers occur everywhere, the demonstration of the rôle of the oligodendroglia is more difficult. However, the retina of the rabbit constitutes the best material to test Rio Hortega's hypothesis that the oligodendroglia either directly secretes myelin or else takes an important part in the elaboration of this substance. This is so because in some areas in the retina of this animal the nerve fibers are all medullated, while in others only nonmedullated fibers are found. The medullated nerve fibers are always surrounded by the prolongations of the oligodendrocytes. No oligodendroglia exist beyond the areas of myelinization. The only interstitial elements found in those portions of the retina that lack myelinated fibers are the cells of Mueller, the astrocytes and the microglia. Likewise, these are the only glial cells found in the normal human retina and in the retinas of those animals in which myelinated fibers are not normally present.

As already stated, there is no lamina cribrosa in the eye of the rabbit. The penetration of oligodendroglia in the retina is, therefore, not opposed by any barrier. As shown by Rio Hortega, the young oligodendroglia cells (oligodendroblasts) migrate passively as the result of repeated divisions. Since the oligodendroblasts occur between fibers, they extend along the path of minimal resistance and hence form lineal series. In the course of development these cells finally reach the retinal portion of the optic nerve. In man and in those animals that have a lamina cribrosa, it is evident that this structure will oppose the entrance of the oligodendroblasts into the retina. However, it is conceivable that retardation in the formation of the lamina cribrosa or a defect due to faulty development might allow the passage of variable numbers of oligodendrocytes. Once within the retina, the oligodendrocytes would perform their normal function, i. e., to secrete or assist in the elaboration of myelin around the axons of the ganglion cells. As a result, one would then have the anomalous appearance of medullated nerve fibers in the retina.

CONCLUSIONS

1. In the retina of the rabbit and extending from the disk in the horizontal meridian there are elongated areas of myelinated nerve fibers. Between the fibers there occur numerous nuclei identical with the nuclei observed in the rows of glial elements in the optic nerve.

2. The silver carbonate technic (for oligodendroglia) shows that the cells found among the myelinated fibers of the retina are definitely oligodendroglia and that the prolongation of these cells are wrapped around the myelinated fibers.

3. Oligodendroglia cells are absent in those portions of the retina lacking myelinated fibers.

4. The silver carbonate method shows that the internal limiting membrane of the retina is not a continuous structure but is a very close meshwork formed by fine branches of processes of the cells of Mueller and the astrocytes.

5. The coexistence of myelinated fibers and oligodendroglia in the retina of the rabbit lends strong support to Rio Hortega's hypothesis that the oligodendrocytes are either primarily concerned in the secretion of myelin or assist in the elaboration of this substance.

Clinical Notes

A NEW AUTOMATIC TREPHINE*

AARON S. GREEN, M.D.; LOUIS D. GREEN, M.D., and MARTIN I. GREEN, M.D.,
SAN FRANCISCO

In a paper on glaucoma published in the ARCHIVES in March, 1930,[1] we stressed the points that in our opinion were the principal factors underlying success in trephining for glaucoma: the avoidance of pressure on the globe and elimination of traction on the iris. The amount of pressure depends on the type and degree of sharpness of the trephine

The new automatic trephine.

as well as on the skill of the operator. In order to cut through the corneoscleral tissue with the ordinary trephine, considerable pressure must accompany the rotary motion produced by the fingers. This manipulation also produces wobbling of the globe with a consequent increase in the amount of trauma.

These disadvantages are practically eliminated with a properly designed automatic trephine. The speed with which the blade revolves reduces pressure to a negligible minimum, and the smooth and uniform revolution of the blade prevents wobbling.

We experimented with numerous models before we perfected one that proved to have the proper shape, speed and weight. If the shape and weight are not correct, they cause a cramped position of the fingers which interferes with the delicacy of touch necessary for the proper performance of the operation. The speed must be right, because if too slow, the aqueous is permitted to seep through before the necessary amount of iris has ballooned out to do the iridotomy, and if too rapid,

* Submitted for publication, May 18, 1931.

1. Green, A. S.; Green, L. D., and Green, M. I.: Glaucoma: Factors Underlying Success in Trephining, Arch. Ophth. **3**:297 (March) 1930.

the ballooning of the iris will be too sudden for the proper performance of the iridotomy. The instrument as finally designed is light and is shaped and held like a pen, and the rate of speed is regulated to what our experience has shown is just right.

The tip of the forefinger rests lightly on the trigger which on gentle pressure releases the rotating mechanism. The ease with which this is done permits the surgeon to place the trephine blade in proper position for the operation without danger of displacing it and perforating the flap, and yet producing minimum pressure on the eyeball. The cutting edge of the blade is superlatively sharp and has a small shoulder slightly less than 1 mm. from the edge, which prevents the blade from entering too deeply into the eye. The blade comes in two sizes, 1.25 mm. for the high tension eye and 1.75 mm. in diameter for the low tension eye. We have been using this instrument since March, 1930, with most satisfactory results.

STERILIZATION OF INSTRUMENT

The body of the instrument is sterilized by applying to its surface pure compound solution of cresol on a cotton applicator, care being taken that the solution does not enter the interior of the instrument. The blade is kept constantly in this solution and when ready to be used, the solution is removed by wiping with alcohol. It is then dried by drawing through it, from the dull to the sharp end, a darning needle threaded with yarn, in this way avoiding damage to the sharp edge.

The instrument can be obtained from the Pacific Optical Company, San Francisco.

Ophthalmologic Review

THE NORMAL VITREOUS HUMOR

A CRITICAL STUDY *

ARTHUR M. YUDKIN, M.D.
NEW HAVEN, CONN.

A great deal has been written about the vitreous humor, but its true normal structure and composition are still unknown. The introduction of improved methods of focal illumination for the study of the vitreous body in situ and the results obtained by workers in other fields on the fluids of the body—the blood, aqueous humor and cerebrospinal fluid—have spurred the ophthalmologist to investigate the nature of the structure of the vitreous body.

ANATOMY

Long before Galen's time, the vitreous was considered as a humor that gave the eyeball its shape, but it was Galen who described it as a substance that was thicker and clearer than blood, had the appearance of molten glass and was colorless. There was less moisture in the crystalline humor than in the vitreous. The crystalline humor was derived from the vitreous, which in turn came from the structure surrounding it, and the latter was a process sent down from the brain.

A better knowledge of the anatomy and histology of the eye revealed the vitreous body as an organized tissue having a cellular framework (Demours, Zinn), so constructed that its meshes enclosed considerable fluid. When the tissue was preserved by fixatives, it was noted that a cortex and a nucleus were present. In the former, concentric layers of varying thicknesses were found, which began at the ora serrata and surrounded the nucleus like an open cup. In the latter, running from the anterior to the posterior end, there was a fissure with many ramifications; this was considered as the rudiment of the canal of Cloquet. When the vitreous was hardened by certain reagents, onion-like layers were visible in the cortex (Hannover), and instead of a fissure like the one noted, orange-like, radiating striations were found (Brücke). Not all contemporary observers concurred in this opinion; Bowman

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* From the Section of Ophthalmology, Department of Surgery, Yale University School of Medicine.

believed that these structures were artefacts produced by the reagents used. Hannover described the presence of a hyaloid membrane, which he considered a cuticular structure that was necessary to prevent the escape of vitreous. On the other hand, some investigators (Henle and Iwanoff) believed that the gelatinous substance was surrounded posteriorly by the limiting membrane of the retina and anteriorly by the posterior layer of the zonula and the capsule of the lens, and that there was no real hyaloid membrane. When such a membrane was found, it proved to be the separated internal boundary membrane of the retina. Therefore, when the tissue examined was not very fresh, the limiting membrane was apt to be removed with the vitreous. (This conception is still held by many ophthalmologists.) The scales that were observed on the outer surface of the supposed hyaloid membrane and were considered by many (Hannover and Finkbeiner) as epithelium were probably nothing more than remains of the torn-off ends of the radiate fibers (Henle and Iwanoff).

A zonula was described which began just beyond the ora serrata, somewhat in front of the equator, as a layer of very fine filaments running in a meridional direction, some of which were followed into the vitreous (Henle) and throughout their course were united into a layer of tissue by a homogeneous cement. This layer of tissue was intimately united to the ciliary region of the retina, as well as to the hyaloid of the ciliary processes. At the ciliary body it divided into two folds, of which the posterior one spread to the posterior and the anterior one to the anterior half of the capsule and here united with it. Thus, between the two folds of the zonula and the margin of the capsule was formed the canal of Petit, which, however, like the serous cavities, was closed during life, as the two folds of the zonula appeared to rest on each other (Henle). These views were crystallized by Schwalbe when he compared the vitreous body to a sponge that was saturated with water. Kessler and Keibel did not subscribe to this idea.

Later, Salzmann described the vitreous body as slightly adherent to the papilla, to the inner surface of the retina and more firmly to the ora serrata and the ciliary epithelium in a zone about 1.5 mm. in width immediately adjacent to the ora serrata. He called this area the base of the vitreous. As a whole, the vitreous body resembled a flattened sphere sagittally, and in front presented a round della (patellar fossa). The posterior and the lateral surfaces of this body represented the mold of the retina, and the patellar fossa in front was the negative impression of the posterior surface of the lens. The transition from the fossa patellaris to the outer convex surface of the vitreous presented a low wall, on the back of which the posterior half of the corona ciliaris appeared in an undisturbed topographical relationship and received the

shallow radial impressions of the ciliary processes. The anatomic picture presented by Salzmann is the impression most contemporary ophthalmologists have of the vitreous humor.

HISTOLOGY

A more detailed study of the histologic appearance given by Salzmann also has its advocates (Szent-Cyörgyi and Fuchs). It reveals that the so-called framework of the vitreous expands out from the base of the vitreous. The posterior border layer is thickest immediately at the ora serrata; further backward it is usually thinner, for the innermost layers constantly turn off into the interior of the vitreous. In this manner, the posterior border layer finally breaks up entirely. It is in firm union with the membrana limitans interna retinae opposite the entrance of the optic nerve in the area of Martegiani. The anterior border layer is much thinner than the posterior layer and maintains this thickness unchanged to the border of the lens. It then thins out in the territory of the lens, and in the middle of the patellar fossa reaches a minimum without disappearing completely. As in the posterior border layer, the thinning is produced by a breaking off of the fibers into the body of the deeper layers. The most important difference, however, between the posterior and the anterior border layer is that the posterior border layer is connected with the membrana limitans interna retinae throughout, whereas the anterior border layer is separated from the analogous membrana limitans interna ciliaris by the posterior chamber. The separation is not complete everywhere, for in places the posterior chamber is crossed by delicate extensions of the vitreous. Wieger reported a structure (ligamentum hyaloideocapsulare) of the vitreous which is united to the capsule of the lens. This structure is prominent in the eye of the ox. Not all observers agree with the conception that has been outlined. Kessler believed the vitreous to be a homogeneous transudate without any structure. Cohenheim, Scalinci and Duke-Elder considered the vitreous to have the properties and structure of a gel.

SLIT-LAMP EXAMINATION

In view of the transparency and the delicacy of the vitreous structure, many difficulties were encountered in its examination. The slit-lamp, introduced by Gullstrand, immediately opened up a new approach to the solution of the problem. The descriptions of the vitreous body recorded by many observers are similar, but the interpretations of the observations are not the same. They found that between the lens and the vitreous humor there is a definite space, and directly posterior to this is a wall of waves. The crests of the waves which appear to be white, may be vertical, horizontal or oblique. The most anterior waves

may have one direction, and the succeeding layers may be arranged at other angles. Between these waves there are fibers of various lengths, contours and thicknesses. The fibers are frequently of uneven surface, giving the impression of the presence of white nodes, which are attached to them (Bedell). The undulations often appear as a very thin, white, gossamer curtain, attached above and below, having innumerable folds or ridges.

Gullstrand, Erggelet, Koeppe, Koby and Vogt have described the vitreous as a framework or scaffolding, hanging curtain-like in undulating folds, moving with each movement of the eye; the folds appear to be made of fibers and strands arranged in interconnecting bundles in an ever-changing but basically stable pattern, and between them are optically clear spaces filled with a clear transparent fluid.

ULTRAMICROSCOPIC EXAMINATION

A more detailed study of the vitreous humor was made by means of the ultramicroscope by Baurmann, Comberg and Heesch. Duke-Elder confirmed their observations, and pointed out the advantages of using this method. He found that when the fresh vitreous is examined under the ultramicroscope, it is at first optically empty, but soon innumerable, minute micelles appear in the beam of light. They are about 30 microns long and 3 microns thick; after standing for some time, they become restless and begin to flit about, until eventually a typical brownian movement is seen. The appearance of the fibrils then changes; they break up into separate particles somewhat resembling strings of beads, and finally dissolve into separate dots. The central part of the vitreous is found to be occupied by long, thin, filmy micelles arranged loosely in an irregular network; nearer the periphery they are shorter, thicker and more closely arranged. There are two regions of the vitreous body which are structurely different from the main mass. The first is the region of the zonula, which is characterized by the coarse nature and close aggregation of its micelles, which have become differentiated to give support to the lens. The second region is that immediately behind the lens, which is prolonged as a narrow tubular extension through the center of the eye to the optic disk, where it again widens out; this region forms the retrolental space anteriorly, the canal of Cloquet in the middle and the area of Martegiani posteriorly. In this area the fibrils are extremely fine and are spaced far apart, and they are separated from the surrounding tissue by a denser structure.

EMBRYOLOGY

The origin of the vitreous humor is still an unsettled question. Several schools of thought have developed since the early recorded

observations of Schöler. He advanced the theory that the vitreous humor is a sort of connective tissue which develops from the mesodermal layers of embryonic tissue. This mesodermal hypothesis was upheld in part by many other observers. Later, Kessler suggested that the vitreous was derived from the embryonic blood vessels as a sort of a transudate. Tornatola pointed out that the mesodermal theory was insufficient to account for the facts, since in most animals the vitreous humor continued to develop after closure of the fetal cleft had severed connection with the surrounding mesoderm and the hyaloid system had atrophied. He assumed that the vitreous was an ectodermal secretion arising from the retinal layer of the optic cup. A study of the development of the eye in amphibia revealed to Rabl that the ciliary region was the main point of origin of the vitreous humor.

Von Lenhossék alluded to the vitreous as a product of the lens, for he observed branching fibrils, arranged in radial and concentric sets, spanning the space between the cone-shaped projections on the deep surface of the plate of the lens and the retina. He believed that these fibrils formed the foundation of the vitreous body. Cirincione considered the ectodermal origin as an early manifestation, for it was present before any vascular mesoderm had grown between the two surfaces, the retina and the lens. After the formation of the capsule of the lens, a mesodermal substance derived from the hyaloid system and an ectodermal substance from the retina invaded the area to become the secondary vitreous. The ectodermal tissue also produced the strong fibers that make up the zonula. This conception was somewhat modified by Druault, for he ascribed the formation of the primary vitreous to a mesodermal origin and the secondary vitreous to an outgrowth of the retina. He described the canal of Cloquet as a primary mesodermal tissue that had been displaced into the axis of the eye by the secondary ectodermal outgrowth of vitreous from the retina.

The third school considers that the vitreous humor is formed from three types of cells: the neural ectoderm, surface ectoderm and mesoderm. The part they play in the process of formation depends on the time and the animal. Wolfrum, von Szily, Froriep and Mann are advocates of this opinion. Mann summarized in a book all of the work that has been published previously on the vitreous. Her conception of the status quo of the origin of the vitreous is as follows: The primary vitreous is formed from the ectoderm of the plate of the lens and the optic vesicle, with a secondary ingrowth and incorporation of a vascular mesoderm. This stage ends with the secretion of the hyaline capsule of the lens, which prevents the lens from taking any further part in the production of vitreous; the secondary vitreous is derived from a fresh outgrowth from the inner layer of the optic cup and the periph-

eral portion of existing vitreous by atrophy of the vasa hyaloidea. Then a line of condensation appears, separating the central vascular remains of the early vitreous from the peripheral vascular secondary vitreous to form the canal of Cloquet and the capsula perilenticularis. The tertiary vitreous is formed from the neural ectoderm of the ciliary region, and the differentiation of this forms the fibers of the zonula.

Thus the adult vitreous is practically entirely derived from the neural ectoderm of the inner layer of the optic cup, and only that portion lying immediately behind the lens and in the region of the canal of Cloquet has in it any elements derived from the surface ectoderm (lens) or mesoderm.

To these three embryonic periods of the development of the vitreous Mann added a fourth, which covers the first three or four years after birth. The changes that occur during this period are concerned solely with the relationships of Cloquet's canal and the anterior end of the hyaloid artery. At birth, Cloquet's canal extends horizontally backward from a point a little below and to the nasal side of the posterior pole of the lens to the optic disk. The extreme anterior end of the main trunk of the hyaloid artery extends horizontally backward from the capsule of the lens along the first part of the canal. After birth, further atrophy involves this vascular remnant, and it gradually drops until it hangs down perpendicularly from the lens. It also becomes curled into a spiral form. At the same time the walls of Cloquet's canal become lax, and the whole structure tends to sag down so that its anterior open mouth, instead of pointing straight backward, comes to lie well below the posterior pole of the lens and finally on a level with the lower border of the dilated pupil. It is thus not obvious in the living eye in the upright position, though the backward slope of the face of the vitreous seen with the slit-lamp is an indication of its continued presence in the lower part of the eye. Its walls remain extremely slack, and in the adult it can easily be made to float up and assume its embryonic position for a short time by appropriate movements of the head and eyes.

CIRCULATION

The question has often been asked whether there is definite circulation in the vitreous. Several observations have been recorded that would definitely indicate the presence of such a movement. Chemicals that were injected intravenously were observed in the ciliary region and choroid, and traces of the substance were later found in the posterior layers of the vitreous near the optic nerve. Leboucq showed that when masses of india ink and oil were injected into the vitreous they remained unaltered for a long period. Duke-Elder expressed the belief that instead of a relatively simple stream of fluid passing from the vasculature

of the ciliary body and choroid, the fluid spread from all the vascularized tissues of the eye, more especially from the ciliary body, by diffusion. The point of most ready exit would seem to be by way of the optic nerve head. Behr, in his experiments, failed to find any evidence of such an exit in man.

CHEMISTRY

Many chemical analyses have been made by early investigators, and their results compare well with some of those recently published. In the last decade, however, more complete studies have been recorded on this tissue. The methods that have been employed are considerably finer and more accurate. Jess, Cohen and his collaborators, Tron and many others have contributed their observations during this period. More recently, Duke-Elder published an exhaustive and painstaking monograph on the vitreous entitled "The Nature of the Vitreous Body." A similar study is in progress in my laboratory. Some of the results are incorporated in this paper.

Most of the chemical statistics on the normal vitreous humor are those that have been obtained by analyzing the material found in the healthy eyes of normal animals. As yet a complete study of the vitreous humor of man has not been published. The question of whether the results obtained from animal tissue can be used as a basis of understanding of the human vitreous often arises. It is my impression that a thorough knowledge of the nature of the vitreous humor of animals leads to a better conception of the human tissue.

When analyzed chemically by modern methods, the vitreous body reveals substances that are found in the blood and the surrounding cellular tissue. The ingredients that probably find their way into the vitreous humor from the blood stream are: albumin, globulin, immune bodies, urea, amino-acids, uric acid, creatinine, lactic acid, sugar, sodium, potassium, calcium, magnesium, chlorides, phosphates, sulphates and substances that are extracted by ether and alcohol. The ingredients coming from the surrounding tissue are the mucoproteins and a substance described by Duke-Elder as "residual protein," lactic acid and some ether and alcohol extractive bodies, mainly cholesterol.

The accompanying table contains the ingredients found in the vitreous humor of the horse (Duke-Elder) and of the ox (Cohen and his collaborators).

Proteins.—The table reveals a number of interesting facts. There is approximately the same amount of solids in the aqueous and vitreous humors; yet the latter has a jelly-like consistency, whereas the former is a fluid. Jess, Ascher and Gebb found that the content of protein in the vitreous is approximately equal to that of the aqueous, whereas Dogiel, Wessely and Franceschetti and Wieland noted that there is less

protein in the vitreous than in the aqueous. When the technic of chemical analyses has been carefully regulated, the fresh vitreous humor is found to be somewhat richer in protein nitrogen than the aqueous, and still richer when the fluids of the eye are examined as postmortem material. The nature of the protein in the vitreous seems to differ somewhat from that in the aqueous humor. The albumin and globulin in the vitreous humor, however, resemble those in the aqueous and the blood serum (Cahn, Boë, Giacosa and Duke-Elder), but the other proteins do not.

In addition to the foregoing proteins, Mörner described a protein which he called "hyalomukoid." After experimenting with it, he noted

The Chemical Composition of the Vitreous Body

	Horse		Ox	
	Vitreous	Aqueous	Vitreous	Aqueous
Water	99.6813	99.6921	.....	.....
Solids	1.1087	1.0869	0.0111	0.0114
Total nitrogen	0.0301	0.0268	0.0227	0.0201
Nonprotein nitrogen	0.0264	0.0236	0.0157	0.0164
Protein nitrogen	.....	.....	0.0069	0.0035
Protein	0.0652	0.0201	0.0393	0.0221
Albumin	0.0077	0.0078	.....	.....
Globulin	0.0115	0.0123	.....	.....
Fibrinogen	traces	traces	.....	.....
Immune bodies	traces	traces	.....	.....
Ferments	traces	traces	.....	.....
Fats	0.0070	0.0040	.....	.....
Cholesterol	0.0005	traces?	.....	.....
Urea nitrogen	0.0290	0.0280	0.0099	0.0085
Amino-acid nitrogen	0.0300	0.0290	0.0018	0.0055
Uric acid	.....	.....	0.0028	0.0017-0.0041
Creatinine	0.0010	0.0020	0.0011	0.0013
Creatine	.....	.....	.....	.....
Lactic acid	0.0190	0.2050	0.0148	0.0189
Sugar	0.0973	0.0983	0.0039	0.0036
Sodium chloride	.....	.....	.....	.....
Sodium	0.2731	0.2787	0.0301	0.0316
Potassium	0.0192	0.0189	0.0279	0.0200
Calcium	0.0068	0.0062	0.0079	0.0074
Magnesium	0.0020	0.0026	.....	.....
Chlorides	0.4168	0.4371	0.0678	0.0688
Phosphorus	0.0031	0.0033	0.0002	0.0054
Sulphur	0.0062	0.0061	0.0004	0.0050-0.0074

that it produced a reducing sugar on hydrolysis. Later, Levene analyzed the mucoprotein that he observed in the vitreous humor and gave it the formula $C_{26}H_{44}O_{29}N_2S_2B_2$, and labeled it "mucoitin sulphuric acid B." Duke-Elder elaborated on this work and suggested that the mucin-like ingredient is probably, in a measure, responsible for the optical function of maintaining the transparency of the vitreous humor. He substantiated this opinion by assuming that the presence of this protein in the umbilical cord and cornea performs a like duty.

Another ingredient described as "skin" and "scaffolding" by Cahn, Mörner and Jess is found in the vitreous humor. Duke-Elder designated this substance as "residual protein." I have also examined this protein-like material and have found it to be as described by Duke-Elder,

a sticky, gummy substance which, after careful washing and drying, resembles a dry and horny piece of gelatin. It does not dissolve in water, but dissolves to a slight degree in acids and to a very marked degree in alkalis. It completely and readily dissolves on boiling with either acids or alkalis. It is not digested by pepsin and trypsin. Duke-Elder gave the composition of this substance as follows: carbohydrate, 44.46 per cent; hydrogen, 6.41 per cent; nitrogen, 12.20 per cent; ash, 3.82 per cent; including phosphorus, 0.12 per cent, and sulphur, 0.675 per cent. He assigned to this protein-like substance the power of forming a gel.

It is evident from the literature that the distribution of immune bodies in the vitreous depends on physical conditions. These substances seem to enter and leave the eye in very minute quantities. It has been pointed out that in highly immunized animals, when their concentration in the blood is raised, they may be present to a considerable extent, and the concentration is increased in conditions in which the permeability of the vessels is increased.

The vitreous humor has small amounts of alcohol and ether—extractable substances. Traces of cholesterol have been found in the vitreous body. The concentration of nonprotein nitrogen in the vitreous humor is approximately the same as that of the aqueous humor in the ox, hog (Cohen, Killian and others), horse (Duke-Elder) and dog (Yudkin). A survey of the table discloses the amount of nonprotein nitrogen in the fluids. There is nothing outstanding in this group.

Sugar.—Most observers have found that the concentration of sugar in the vitreous is lower than that in either the aqueous or the whole blood drawn at the same time. The amount is usually the same in the two eyes. If the aqueous and vitreous remain in undisturbed contact with the ocular tissue after death, the sugar disappears in from twenty-four to forty-eight hours, but when the fluid is isolated, the content of sugar is usually greater.

Experiments show that the diffusion of sugar from the anterior into the posterior chamber takes place very slowly, if at all, at normal intra-ocular pressures. Adler found that the concentration of sugar is not uniform throughout the entire vitreous, and that the posterior layers in contact with the retina contain less sugar than the anterior layers. He suggested that the low concentration of sugar in the vitreous is probably due to the marked glycolytic power of the retina. This is corroborated by the work performed by Warburg and his school. Cohen, Killian and Metzger suggested that the lessened concentration of sugar in the vitreous is probably compensated for by the increase in the concentration of chlorides.

It is evident from the statistics that there is a slight difference between the amount of cations (sodium, potassium, calcium and magnesium) and the amount of anions (chloride, phosphates and sulphates) in the vitreous humor. The cations are found in less concentration than the anions. Duke-Elder pointed out that the constituents that are probably derived from the blood are present in proportions comparable to those found in the aqueous humor.

PHYSICAL PROPERTIES

The specific gravity of the vitreous body in man is 1.0053 (Jess); in the horse at 15 degrees, 1.0079 (Duke-Elder), and in the hog, from 1.00 to 1.014 (Felchlin). The refractive index in man varies from 1.3395 to 1.3315; in the horse, from 1.33302 to 1.3363; in the ox, from 1.33304 to 1.3348, and in the hog, from 1.3333 to 1.3360.

From a study of the optical properties of the vitreous body, Abé concluded that it has two iso-electric points, p_H 3.8 and 9.4. Cavazzani, Scalinci and Dieter found that the vitreous filtrate has a greater viscosity than the aqueous humor. Cohen, Killian and Kamner, measuring the vitreous at various temperatures, showed that it approximates that of the blood serum. The few records of conductivity of the intra-ocular fluid disclose the fact that that of the aqueous humor is slightly greater than that of the vitreous filtrate.

The osmotic pressure has been measured by two methods. The results of these determinations show that there is little, if any, difference between the osmotic pressures of the aqueous humor and the vitreous filtrate. The conductivity of the vitreous humor varies somewhat from that of the other body fluids, but most workers are in accord that the aqueous is slightly more electrically active than the vitreous. From a study of the variations in the vapor pressure of the vitreous body, Duke-Elder concluded that the vitreous is probably a reversible, elastic gel, showing no "opacity point," and that it is hydrophilous.

CONCLUSIONS

From the present review and my experience in the laboratory with the preparation and analyses of the fresh vitreous humor from the eyes of the steer, hog and dog, I find that the tissue is a jelly-like substance that is not easily broken up into fluid and its accompanying "skin," "scaffolding" or "residual protein." It is slightly adherent to the inner surface of the retina and more firmly attached to the ora serrata and the ciliary epithelium in a zone varying from 1 to 2.5 mm. in width, immediately adjacent to the ora serrata. It is sometimes difficult to remove the vitreous humor without taking the ciliary epithelium with it. In the ox the vitreous seems to be attached also to the capsule

of the lens. When examined with the slit-lamp, the enucleated vitreous humor is an exact negative mold of the whole vitreous cavity. Because of the difficulty encountered in keeping the protein of the vitreous from denaturing, I have been unable to satisfy myself that the "mucoitin sulphuric acid B" gives the vitreous its transparency, and that the "residual protein" is responsible for the gel of the vitreous humor.

It is my impression that there is still some other ingredient that is probably responsible for the jelly-like nature of the vitreous. This review discloses the fact that there is still much to be learned about the vitreous humor.

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News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Wills Hospital.—The Wills Hospital of Philadelphia has sold the site which it has occupied since its founding one hundred years ago, and has obtained a splendid location on which it will erect an eight story, two hundred bed structure with ample accommodations for outpatients. For the first time in its history the hospital will care for private room patients. Well organized associated clinics, conducted by specialists in diseases of the nervous system, nose, throat and teeth, as well as those demanding the attention of internists, will enable the hospital to carry on all phases of its work within its own walls. Twenty-five thousand outpatients are now cared for annually. The new building will be ready for occupancy in the autumn of 1932.

Fourteenth International Ophthalmological Congress.—1. The Fourteenth International Ophthalmological Congress will take place in Madrid at the beginning of April, 1933. The principal subjects to be discussed at this congress are: (*a*) *Tuberculosis of the Iris and Corpus Ciliare:* Modern Methods of Treatment, Dr. E. V. L. Brown, University of Chicago, 950 East Fifty-Ninth Street, Chicago; Pathological Anatomy, Prof. Dr. J. Igersheimer, Brentanostrasse 1, Frankfurt a. Main, Germany; Diagnosis and Differential Diagnosis, Dr. Henri Lagrange, 5 avenue Daniël Lesueur, Paris, VII, France; (*b*) *Detachment of the Retina:* Etiology, Dr. H. Arruga, Aragón 271, Barcelona, Spain; Medical Treatment, Prof. Dr. G. Ovio, Viale Castro Pretorio 66, Rome, Italy; Operative Treatment, Prof. Dr. A. Vogt, Rämistrasse 73, Zürich, Switzerland. Authors of papers connected in any way with one of these two subjects are kindly requested to send these papers to the reporters in question. 2. All communications to the congress shall essentially refer to the two subjects named. There will, however, be reserved two or three meetings for free communications, including one demonstration meeting. The number of lectures will be limited, both with regard to those that correspond with the principal subjects and with regard to free communications. For this reason papers must be received by the secretary of the International Ophthalmological Council by Dec. 1, 1932, at the latest. In order to secure the best selection from all papers sent in, an international committee of ophthalmologists will review all communications. After that, authors of communications will be informed whether their paper may be read or not. 3. With regard to reports drawn up for the Thirteenth International Ophthalmological Congress at the request of the International Ophthalmological Council, the complete commission of reporters will take decisions during the Fourteenth Congress. These decisions will be communicated to the ophthalmologists individually, to ophthalmologic societies and, if necessary, to the governments with a view to practical application. Hence, all

ophthalmologists who have hitherto failed to make any observations on the conclusions of the reports named (to be found in the Proceedings of the Thirteenth Congress, vol. 4) are earnestly requested to address any observations or comments they wish to make, before May 1, 1932, to the secretary of the International Ophthalmological Council. 4. The reporter on the subject "Pathological Anatomy of Tuberculous Iridocyclitis," Prof. Dr. J. Igersheimer, requests all those who possess special preparations or slides on cases of tuberculous iridocyclitis to forward them to his address for perusal. 5. Oculists who wish to develop their professional knowledge abroad, either by a short visit to a foreign clinic or by a longer stay as a volunteer, may apply to the secretary of the International Ophthalmological Council, who will try to find a place that will suit them. 6. In the year 1929, during the Thirteenth International Ophthalmological Congress at Scheveningen, the International Ophthalmological Council decided to start an ophthalmologic library. Most of the ophthalmologic periodicals have already been collected there. The library is established in Leiden. The object is to collect there everything that will be printed in the domain of ophthalmology. Our colleagues are therefore kindly requested to address an offprint or copy of their publications (viz., reviews in periodicals, books, theses, etc.) to the librarian of the International Ophthalmological Library, Dr. J. Kroon, Stationsweg 25, Leiden, Holland. Further particulars on the working method of this library will follow in due course.

E. MARX, M.D., Rotterdam, Holland,
Secretary, International Ophthalmological Council.

Middlemore Prize.—The Middlemore Prize, a check for 50 pounds and a certificate, will be awarded this year to the author of the best essay on "Sympathetic Ophthalmia Before and After 1914." Essays must reach the Medical Secretary, British Medical Association House, Tavistock Square, London, W.C.1, not later than Dec. 31, 1931. They must be signed with a motto and accompanied by a sealed envelop marked with the same motto and containing the candidate's name and address.

DEATHS

It is with much regret that we learn of the death of Prof. Otto Haab in Zurich. A full notice will appear in the next number of the ARCHIVES.

Obituary

PROF. K. K. K. LUNDSGAARD
1867-1931

The sad death of K. K. K. Lundsgaard, the eminent Danish ophthalmologist, on August 30, in his sixty-fourth year, will be received with deep sorrow by ophthalmic surgeons the world over.

A very painful cancerous metastasis in the first lumbar vertebra had confined him to his bed for the past four months, and pneumonia released him from his sufferings.

Lundsgaard graduated in 1893 in Copenhagen. During the following ten years he studied surgery, bacteriology and ophthalmology, both at home and abroad. This broad medical education, combined with his appointment as oculist to the Finsen Medical Light Institute for twenty-three years, has put its stamp on Lundsgaard's many scientific papers, which numbered about seventy.

At the Finsen Institute, he had ample opportunity to study tuberculosis of the conjunctiva, on which he has written many papers, devoting himself particularly to clearing the differential diagnosis between the primary and the secondary forms of this disease. He introduced Finsen treatment in ophthalmology, and invented ingenious instruments for its use. He early recognized the possibility of general light treatment.

His interest in bacteriology was foremost, and in 1897 he received the University Gold Medal for a treatise on "Ophthalmoblennorrhoea Neonatorum." In 1900, his dissertation for the degree of M.D. was on "Bacteriological Studies in Conjunctivitis."

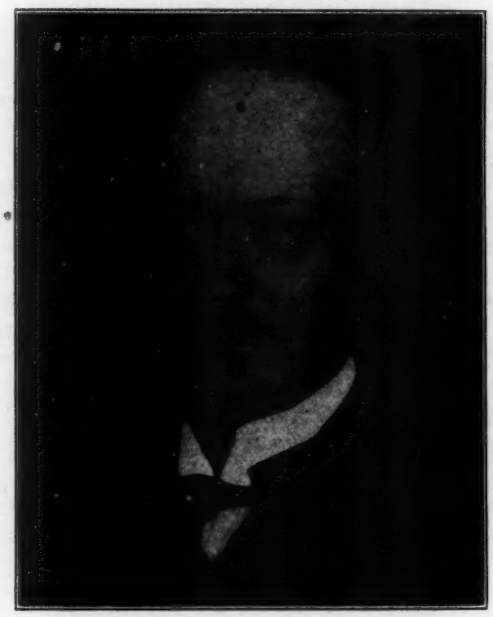
When he competed for the professorship of ophthalmology in Copenhagen University in 1925, he discussed the pneumococcus in its relation to ophthalmology; and when invited to deliver the Doyne Memorial Lecture, at Oxford, in 1927, he spoke on this topic.

Lundsgaard has made valuable contributions to nearly all clinical fields of ophthalmology. I shall confine myself to mentioning "Sterilizing of the Conjunctiva Before Operations," "Fistulizing Operations for Glaucoma," for which he invented a very practical type of trephine, "Erythropia in Alcoholic Hemeralopia," and "Transitory Alterations in Refraction."

Lundsgaard took a very keen interest in the history of ophthalmology, particularly as it concerned spectacles and the operation for cataract; he possessed a very interesting collection of old spectacles and ophthalmologic prints.

The burning love that Lundsgaard bore to his work from the beginning to the end made him the leader in Danish ophthalmology during the last thirty years, and he participated very keenly in the scientific life both in Scandinavia and in other countries.

In 1900, he founded the Ophthalmological Society in Copenhagen. In 1923, he founded the *Acta Ophthalmologica,* whose chief editor he was until his death. He was one of the editors of the *Klinische Monatsblätter für Augenheilkunde,* and a member of the Oxford Ophthalmological Congress, where he often read papers. He attained the great honor to be elected president of the Conseil International



PROF. K. K. K. LUNDSGAARD
1867-1931

d'Ophthalmologie, but his death deprived him of the honor of presiding at the coming International Congress, in Madrid.

Before Lundsgaard became professor of ophthalmology, he was attached to several hospitals in Copenhagen. He has always been highly appreciated by his patients, as he was an unusually skilful oculist and a splendid operator; but first of all he was a true gentleman and friend.

The many colleagues who have known Lundsgaard personally will miss him all their lives, and so will his widow, his daughter and his two sons.

C. F. Bentzen.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

DIAGNOSIS OF VITAMIN A DEFICIENCY (KERATOMALACIA) BY BACTERIOSCOPY OF THE CONJUNCTIVA. ARNOLD PILLAT, Ztschr. f. Augenh. **73**: 244 (Feb.) 1931.

A case history is given of a child, 2 months old, who showed a clinical picture of keratomalacia in the left eye and an apparently normal right eye. The examination of the corneal tissue revealed a like bacterial flora in both eyes, notwithstanding the normal appearance of the right. Pillat asserts that it is possible to detect early stages of keratomalacia by the superabundance of the saprophytic organisms, particularly the xerosis bacillus in the epithelium of the cornea. He observed that when the cornea of a normal eye is scraped in the usual manner with a platinum loop, few or no cells are rubbed off; whereas, in keratomalacia the cells are plentiful in the scraping. The epithelial cells are distorted and stain poorly. There are very few lymphocytes or leukocytes in the scrapings. The author believes that the damage to the eye is produced primarily by a degenerative process and not by an inflammation and that the proliferation of the germinal layer is not increased by irritation but by the proliferation of the saprophytes on the degenerative tissue. This picture of keratomalacia is in accord with that published by the abstractor in *Archives of Ophthalmology* (**52**: 416 [Sept.] 1924).

A. M. YUDKIN.

Congenital Anomalies

THE EMBRYOLOGIC DEVELOPMENT OF CANALICULI LACRIMALIS IN FACIAL FISSURE. HOLGER EHLERS, Acta ophth. **8**: 305, 1930.

The author describes a case of bilateral oblique facial fissure with upward palpebral coloboma and considers the theories proposed for this anomaly. The embryologic relations involved in the formation of the face and the premordial development of the nasolacrimal ducts are discussed. From this study he concludes that the premordial development of the nasolacrimal canals starts from the very point where the primary oblique facial fissure has its origin, and the canaliculi lacrimalis originates from the upper end of the ducti nasolacrimalis premordium. He notes that Fleischner believes that both canaliculi in mammals are derived from the upper constricted end of the duct and grow into the protuberance of the eyelids, until they come in contact with the epithelium of the lid margin. The case in question corroborates the alleged origin of the canaliculi from the lacrimal tissue rather than from the epithelium of the margin of the lid.

A. M. YUDKIN.

Cornea and Sclera

KRUKENBERG'S SPINDLE. M. H. POST, Am. J. Ophth. **13**: 987 (Nov.) 1930.

The author reviews the literature on Krukenberg's spindle, discusses the etiology and concomitant ocular diseases and reports two cases. He calls attention to the fact that practically all observers reporting one case have reported several, suggesting that the condition is not so rare as the paucity of reported cases indicates.

W. S. REESE.

BEE STING OF THE CORNEA. C. A. YOUNG, Am. J. Ophth. **14**: 208 (March) 1931.

The author describes the very interesting mechanism of the bee sting. He discusses the chemistry of bee poison, the effects of stings by various insects and gives the data of experimental studies on the effect of bee stings. The primary injury to the eye is very severe. A secondary injury may occur due to retention of the sting. A girl, aged 20, was stung by a bee in her left cornea. The eye showed moderate injection of the bulbar conjunctiva, and the cornea presented a 2 mm. opacity 3 mm. inside the limbus at 7 o'clock. The pupil was two-thirds dilated and inactive to light. Tension was normal. Corrected vision was 6/9. The biomicroscope showed two linear, golden brown structures lying obliquely in the substantia propria, the tip of one being in Descemet's membrane, and the tip of the other just within the anterior chamber. Surrounding the bee stings and anterior to them was an opaque area 2 mm. in diameter. No changes were noted in the iris, anterior capsule or lens proper. The eye pained so that the patient wanted it removed. Four operations were performed, the stings finally being removed by making a keratone incision and leaving the instrument in position until removal was effected. The patient made an excellent recovery.

The material is summarized as follows: 1. Bee stings of the eye cause both a marked local and general reaction. 2. Local reactions include conjunctivitis, keratitis, hypopyon, iritis, atrophy of the iris, anterior capsular cataract, abscess of the lens, perforation of the globe, glaucoma and change in refraction. 2. The general reactions are diarrhea, loss of appetite, disturbance of heart action, fever, delirium and, at times, death. 4. The eye may be injured primarily or may be affected months or years later by a bee sting's working its way through the lid and then injuring the globe. 5. Retention of bee stings in the cornea evidently does cause irritation at times, as shown in the case reported, even though the rabbits experimented on by Huwald showed no ill effects from retention of stings. 6. Bee stings are probably never absorbed by the tissue, one of Dorff's patients having retained a bee sting unchanged for seven years. 7. Flury believes that the active substance in bee poison constitutes a transition between protein-free sapotoxin of animal origin, such as crotalotoxin and ophiotoxin of snake poison, on the one hand, and the poisons of the cantharid group on the other.

W. S. REESE.

INFORMATION ON THE HEREDITARY SYNDROME OF ABNORMALLY BRITTLE BONES, BLUE SCLERAE AND POOR HEARING. C. K. FRIEDBERG, Klin. Wchnschr. **10:** 830 (May 2) 1931.

In 1900, Eddowes, in reporting this syndrome for the first time, advanced the opinion that it was caused by an anomaly of the supporting tissues. Bauer went further and introduced the thought that it was due to some poverty of the mesodermal layers. Other authors have seemingly brought support to this theory. It is Friedberg's contention that from a study of the cases reported in the literature, together with five cases that he briefly reports, but has thoroughly studied, that Bauer's theory may be proved false, and a new hypothesis substantiated.

The author concludes that because (*a*) most of the mesenchymal structures were not affected, and (*b*) often ectodermal structures were affected, Bauer's contention fails. Blue sclerae have been found in many congenital defects of the eyes, both from mesodermal and ectodermal origin. A bibliography accompanies the article. L. L. MAYER.

Experimental Pathology

CRITICAL AND EXPERIMENTAL STUDIES OF THE SO-CALLED "INFRA-RED CATARACT" AND GLASSBLOWERS' CATARACT. H. GOLDMANN, Arch. f. Ophth. **125:** 313, 1930.

From a critical survey of the literature and from experiments of his own the author concludes that the cataract that occurs in rabbits after exposure to infra-red rays is due to absorption of the rays by the iris and secondary conduction to the lens. If the lens is exposed to the rays without an absorbing medium in front of it, the lens remains clear. The occurrence of opacities of the lens can be avoided if a special cooling system is installed around it. The iris of rabbits absorbs 98 per cent of the infra-red rays. A temperature over 55 C. in the iris is necessary to cause cataract.

For the cataract that occurs in glassblowers Goldmann thinks that direct action of rays is out of the question. It is most likely caused by continued exposure to heat rays, which are absorbed by the iris and secondarily conducted to the lens. P. C. KRONFELD.

General Diseases

THE OCULAR COMPLICATION OF ACNE ROSACEA. J. H. DOGGART, Brit. J. Ophth. **15:** 446 (Aug.) 1931.

After enumerating the various ocular complications of acne rosacea found in the literature, the author gives his personal observations. Reference is made to the following ocular complications noted by other observers: blepharitis, chalazia, conjunctivitis, episcleral nodules and keratitis. Iritis is not a complication per se, but can arise from severe corneal complications in this disease.

The author has personal notes of seventy-eight cases of acne rosacea. The age of the patients ranged from 28 to 67, but most were between 35 and 55; 83.3 per cent were women. Sixty-one suffered from ocular complications, but it must be remembered that the great majority of the

seventy-eight were ophthalmic outpatients. Among thirteen patients seen in a skin department, none had ever suffered from ocular complications. Inquiries into diets, indulgences and general health of the patients did not lead to the discovery of any constant group of etiologic factors apart from those of age and sex.

As a rule, the face was severely affected in those who displayed ocular complications, and the onset of a fresh attack of conjunctivitis or keratitis coincided with a return of the facial efflorescence. Some degree of blepharitis with scaly desquamation was observed in all those that showed any ocular complications. Conjunctivitis was a prominent feature in all cases with keratitis, but seldom occurred independently.

Thirty-eight patients had corneal lesions. The first indication was an encroachment of superficial vessels from the ocular conjunctiva on to the periphery of the cornea around all or any portion of its circumference. Later, one or more grayish-white infiltrate appeared at some part of the periphery of the cornea, more often the lower half. Superficial new vessels always accompany the infiltration, and often form characteristic loops. They remain conspicuous even in the quiescent stages of the disease, showing a vigorous flow of blood. The infiltrates and vessels tend toward the center of the cornea. Unstable ulcers may develop. Small depressed scars forming facets were noted in seven cases. Independent infiltrates may develop, and occasionally an interstitial abscess occurred. The deeper layers were seldom affected. A striking feature emphasized by the biomicroscope is the roughening of the cornea.

The treatment employed is detailed.

The author concludes that by reason of its capacity to produce keratitis in a small proportion of cases, acne rosacea may bring about serious disability. Keratitis in this disease causes repeated attacks of pain in the eyes and endangers clear vision. It is exceedingly difficult to cure, and may be almost impossible to relieve. If one system of treatment fails, then others should be tried, in order to shorten the attacks and to increase the intervals of quiescence. A permanent cure is not claimed in any single instance. Treatment by ultraviolet light or x-rays offers a chance of temporary improvement when simple remedies have produced no response.

W. ZENTMAYER.

Two NEW SYMPTOMS IN OXYCEPHALY OR IN THE PREMATURE CRANIOSYNOSTEOSES AND THEIR PATHOGENESIS. D. GOURFEIN, Arch. d'opht. **48:** 112 (Feb.) 1931.

The author calls attention to two new ocular symptoms in this type of malformation. In addition to exophthalmos and optic atrophy, which are the most common ocular abnormalities, he has observed three cases in which bilateral ptosis and paralysis of convergence were present, and abstracts a fourth previously reported by Mirimanoff in 1924. He discusses the theories suggested to account for these conditions and expresses his belief that although they may have a common cause, they are probably not directly the result of the cranial deformity.

S. B. MARLOW.

DIABETES AND THE EYE. MAX ROSENBERG, Ztschr. f. Augenh. **73:** 207 (Feb.) 1931.

Rosenberg classifies his cases of diabetes into two groups: the severe diabetes in childhood and young adult life up to 40 years, and the less severe cases in older persons. He contends that a large percentage of the changes found in the fundi of adults suffering from the disease should be considered as eye disturbances accompanying diabetes rather than produced by it, for the ocular lesions are rarely met with in children, even though their diabetic conditions are severe. With the advent of insulin therapy, other manifestations of metabolic disturbances have become conspicuous. The most outstanding disturbances are paralysis of accommodation and changes in the refractive index of the intraocular fluid. They are not infrequently encountered in patients who have neglected their therapeutic regimen. The prognosis is usually favorable, and the disturbances disappear in from one to two weeks, depending on the adaptation of the body to its new normal metabolism. Ocular palsies have been described in diabetes, but Rosenberg has never observed such lesions. He has never found the so-called retinitis of diabetes in children. He attributes the changes in the fundus to vascular disturbances that are independent of diabetes.

He has noted that cataracts develop in persons with diabetes at a much younger age than in nondiabetic persons. At least this is true when treatment has been neglected. As a rule, cataracts do not disappear when treatment is given. He notes that there is no greater affinity for inflammation and infection, such as conjunctivitis, blepharitis, styes, keratitis and iridocyclitis in diabetic persons than in those with any other internal diseases. Glaucoma is infrequent.

A. M. YUDKIN.

ARTERIAL HYPERTONY AND THE EYE. G. GUIST, Ztschr. f. Augenh. **73:** 232 (Feb.) 1931.

Guist notes that in toxic hypertony large streaks are regularly seen accompanying the arteries, and the small arteries are barely visible. The large and small veins are tortuous but are not of the corkscrew type. Histopathologic sections of the choroid and retina in this type of disturbance show a marked involvement of veins and arteries so that it is difficult to tell them apart. In simple hypertrophy the arterial walls are increased, and the small veins have a thin wall. The blood in the rigid arterial stream under high pressure is forced into the thin-walled veins, and the expansion force of the fluid distends the venous wall, so that the small, almost invisible veins assume a clublike appearance. Histopathologic sections of the choroid and retina show that the arteries have a deposition of hyaline material, and the veins show almost no abnormalities. According to this finding, Guist explains why in primary hypertrophy the corkscrew undulations are present in the small veins and not necessarily in the larger one, whereas in toxic hypertony only moderate tortuosity of both large and small veins is noted.

A. M. YUDKIN.

Injuries

INJURY TO THE EYE BY RADIUM THERAPY. O. BLEGVAD, Acta ophth. 9: 32, 1931.

The author describes the lesion and the treatment employed in twelve cases of neoplasm of the lid. Severe conjunctivitis developed in almost all the cases in which radium was used. The inflammation was probably produced by the rays rather than by the manipulation of the tissues with the lead prosthesis. As a rule, the conjunctivitis cleared up satisfactorily after the termination of the exposure to the radium rays. In several cases an engorgement and tortuosity of the scleral vessels developed. They, however, did not disappear at the end of treatment of the eye with radium. Several types of corneal lesions were noted, but they were not all due to the radium exposure. In a number of these cases the corneal cloudiness was probably due to absence of protection of the cornea by lid tissue, keratitis e lagophthalmo. The corneal lesions, which were due to radium exposure and its protecting lead sheets, healed rapidly when proper treatment was instigated.

Only one case of injury to the iris was observed, and this was accompanied by cataract formation. Several patients showed cataracts, but only four of these can be attributed to the radium exposure. Blegvad believes that the lack of protection was responsible for the lenticular changes. He operated on one of them, but did not encounter the complication of hemorrhage as already reported in the literature. No fundus changes were noted. In one case, a definite constriction of the field was found, whereas the untouched eye showed a normal field. The fundus picture was normal. Urbanek reported a similar occurrence. He is very enthusiastic about the use of radium in the treatment for carcinoma of the lid, for it gives a favorable result if good judgment and care is used in the protection of the surrounding tissue. A. M. YUDKIN.

Lacrimal Apparatus

ISOLATED LACRIMAL CANALICULUS INFLAMMATION (DACRYOCANALICULITIS). F. HERRENSCHWAND, Wien. klin. Wchnschr. 44: 871 (July 3) 1931.

The lacrimal canaliculus seldom participates in the inflammatory processes of the lacrimal passages or the conjunctiva. Usually it is a chronic lacrimal condition that affects the canaliculus, such as is found in old trachoma, etc. The case of dacryocanaliculitis presented is the sixth to be found in the literature. The others have been reported by Elsching, Hortach, Wiltschke, Stock and Theobald.

A man, 64 years old, who had noticed a swelling of the right upper lid near the inner canthus, complained that his vision had been disturbed from childhood by an inflammation of the eye, but was now more distorted because of the tumorous mass. The nasal third of the upper lid was swollen to the size of a cherry seed and fluctuated. The skin of the lid seemed normal here, but was stretched and thinned. The palpebral margin was rounded off in the region of the swelling, and at the same time was turned outward so that a portion of the palpebral conjunctiva could be seen, which appeared very red and thick. The upper lacrimal

punctum gapped open and receded from the eyeball. No secretion could be squeezed out. The bulbar conjunctiva and lacrimal organs were normal. Vision was 6/12. The left eye was normal. A fungus concretion was thought of as being the causative agent. During the attempt to pass a sound, an opening into a saclike structure was made, and a vitreous-like fluid was excreted. Bacteriologic study showed diplobacilli, pneumococci, sarcinae and pyocyanei. The author agrees with Stock that an injury to the mucous membrane of the canaliculus, which thus allows of infection, may be considered to constitute the etiology of the condition. A short bibliography follows the discussion. L. L. MAYER.

Lens

CATARACT AFTER THYROIDECTOMY. R. T. BRANBERGEN, Arch. d'ophth. **48:** 120 (Feb.) 1931.

The case of a woman who had had a thyroidectomy done when she was 34 years old is reported. The day after operation symptoms of tetany appeared, which were controlled by calicum therapy after the failure of parathyroid extract. Four years later, failure of vision was first noted; this continued in spite of treatment until a cataract operation was done at the end of another two years. During this time there had been no spasmodic attacks, nor were there any changes in the hair or nails. Biomicroscopic examination showed the changes to be situated in a thin uniform layer beneath the capsule. These changes consisted in snow flakes and varied colored crystals.

Branbergen refers to cases reported by Greppin, Van Lint, Kneusel, Heine, Kast and Heinonen. In 1928, L. Jacques collected thirty-four cases from the world literature. In his conclusions, Branbergen calls attention to the fact that most of these cases have occurred in regions where goiter, and therefore, thyroidectomy, is common. In some cases there have been two operations. Symptoms of tetany have almost always been manifest early. The biomicroscopic picture is not always the same. Treatment by parathyroid extract was of little benefit in the case reported. S. B. MARLOW.

Methods of Examination

A SIGN IN DIFFERENTIAL DIAGNOSIS OF LESIONS PRODUCING UNILATERAL EXOPHTHALMOS. J. WOLFF, Am. J. Ophth. **13:** 989 (Nov.) 1930.

The author has noted differences in sound by auscultation over exophthalmic eyes. He gives the following conclusions: 1. The nasal respiratory sound and vocal fremitus are intensified over the protruding eye when the lesion causing the exophthalmos is firm and solid (e.g., osteosarcoma, endothelioma). 2. The nasal respiratory sound and vocal fremitus are diminished over the protruding eye when the lesion causing the exophthalmos is soft or contains fluid (e.g., cysts, very vascular tumors). 3. The location of the lesion in the orbit is probably a factor; the sounds are more likely to be influenced, either in the direction of

intensification or diminution, if the lesion is near the nasal orbital wall than if it is distant from it.

The method of testing for the sign is: The tip of a conical stethoscope is pressed gently and straight over the normal eyeball, with the lids closed as in sleep. The patient is asked to breathe deeply and regularly in and out through the side of the nose corresponding to the eye that is being tested, the other naris being held shut, and the examiner notes all the characteristics of the sounds made by the air current as it passes through the nose. The stethoscope is then transferred to the protruding eye for comparison, and this process is repeated several times, until the examiner is convinced of the findings. The proceeding is then repeated while the patient says "one-two-three" or "ninety-nine." It is important, before making the test, to ascertain that both sides of the nose are patent and that there are no extraneous sounds caused by obstructions. In that case the mucous membrane should be shrunken with cocaine or epinephrine, until the air current as heard in front of the nose produces the same sound from each meatus. W. S. REESE.

Ocular Muscles

DIFFERENT CLINICAL VARIETIES OF STRABISMUS AND THEIR SURGICAL TREATMENT. R. ARGAÑARAZ, Arch. de oftal. de Buenos Aires **6:** 127 (March-April) 1931.

The author deals separately with: (1) convergent strabismus, (2) divergent strabismus, (3) vertical strabismus with torticollis and (4) vertical strabismus without torticollis.

Convergent Strabismus: After a brief reference to his toxi-infectious-pathogenetic theory—with localizations in the motor and sensorial centers presiding over binocular vision and with secondary contraction of the opposing muscles—the author states that he favors, as more scientific and as more apt to be followed by a favorable result, a bilateral tenotomy of the interni, freeing the tendon from superficial (conjunctival) and deep (scleral) connections. The best time to operate is after the fifteenth year when the eye is fully developed and permanent conditions have been established. He considers the classic unilateral tenotomy with advancement of the opponent unsound. It should be reserved for exceptional cases, mostly for squints under 20 degrees.

Divergent Strabismus: Three varieties are considered: (1) divergent strabismus from amblyopia, (2) functional divergent strabismus of early childhood and (3) paralytic divergent strabismus. The first variety is due to the lack of a stimulus for binocular vision, the lack of vision of the squinting eye inducing it to take the position of anatomic rest; as a rule, it is under 15 degrees. The second variety is characterized by two special clinical features: (*a*) that once initiated, it does not yield to any medical or optical treatment, and (*b*) that it increases steadily, having no tendency to become stationary. The third form after one year's duration can be subjected to some surgical procedure. The author prefers a double tenotomy of the externi to other procedures. When an advancement of the internus is necessary, he

performs a capsulomuscular advancement with fixation of the eye in convergence by means of three sutures.

Vertical Strabismus with Torticollis: All cases of torticollis demand an examination of the muscular apparatus of the eyes, many being cured by means of an ocular operation. Most commonly the condition is caused by a paralysis of the superior oblique and next in order of frequency by paralysis of the superior rectus. In these cases to avoid diplopia the patient rotates the head on a vertical axis and inclines it to the opposite side. In paralysis of the right superior oblique the head is rotated toward the right and is inclined toward the left. In cases of paralysis of the superior oblique the author tenotomizes the inferior rectus of the other eye; in paralysis of the inferior oblique (rare), the superior rectus of the same side. In paralysis of the superior rectus he tenotomizes the inferior rectus of the same side.

Vertical Strabismus Without Torticollis (Functional Vertical Strabismus): In vertical strabismus due to weakness or lack of a sense of fusion, the necessary nervous stimulus to maintain the ocular axes parallel or to provoke compensatory deviations of the head does not exist. The author performs a capsular advancement in the upper or lower part of the deviated eye. In cases of superior strabismus he excises a vertical piece of the lower conjunctiva and underlying capsule, from 1.5 to 2 cm. in length, commencing at the inferior corneal margin. He then unites the edges of the wound by means of vertically placed sutures. When he does not obtain sufficient effect, he produces a relaxation of the upper part of the capsule by means of the excision of a horizontal strip, 2 cm. in length, with union of the edges of the wound by means of horizontally placed sutures. C. E. FINLAY.

Operations

PLASTIC SURGERY OF THE FACE. A. CUCCO, Ann. di ottal. e clin. ocul. **59:** 272 (March) 1931.

The author reviews the history of plastic surgery, especially that of the periocular region and considers whether such work properly belongs to general surgery, ophthalmic surgery or the so-called "aesthetic surgery." He believes that the ophthalmic surgeon, by virtue of his training in the use of fine instruments for delicate procedures and his knowledge and respect for the important structures of this region, is eminently fitted to carry out such work. A number of procedures are illustrated by cases from the Cirincione Clinic. The author reports two personal cases that illustrate the need of combining a number of procedures to obtain a good result. In one patient, a man with scarring extending over half of the face and marked ectropion, repair was done with a large pedicled flap from the brow and also sliding flaps from the surrounding region. A good cosmetic result was obtained. In a second patient with considerable deformity following malignant pustule, a good result was obtained by sliding flaps. Thiersch grafts are necessary in some cases, but their drawbacks of shrinkage and a tendency to necrosis must be remembered. A bibliography is included. S. R. GIFFORD.

Orbit, Eyeball and Accessory Sinuses

OBSERVATIONS ON THE TREATMENT OF ORBITAL OSTEOMA, WITH REPORT OF A CASE. N. PATTERSON and H. CAIRNS, Brit. J. Ophth. **15:** 458 (Aug.) 1931.

The authors point out that an osteoma of the orbit, aside from its effect on the eyeball and its nerves and on the ethmoidal sinuses, may, as Cushing has shown, in the course of its expansion, project through the floor of the anterior fossa and become adherent to and finally penetrate the dura, giving rise to serious complications. This fact has been responsible for the development of an intracranial method of operation for these tumors.

A single woman, aged 26, gave a history of slowly increasing exophthalmos on the left side extending over a period probably of two or three years. The eyeball was displaced forward and upward without limitations of the movements of the globes. There was no diplopia. On firm pressure backward in the region of the left inner canthus a hard nodular immovable mass could be felt. There was a moderate swelling of the left optic disk. Visual acuity equaled 6/36 partly. There was enlargement of the blind spot with constriction of the upper field in the smaller isopters. The roentgenogram showed the roof of the orbit to be intact.

The osteoma was removed by the naso-orbital route. An incision was made along the inner half of the left eyebrow and down on to the left side of the nose as far as the level of the inner canthus. After the soft tissues had been reflected, portions of the frontal process of the left superior maxilla, nasal and lacrimal bones were removed. Some of the anterior ethmoidal cells were opened, and the anterior portion of the osteoma was exposed. The eyeball was now retracted outward, and the periosteum was separated from the accessible part of the tumor, which was gently rocked in a lateral direction. It was thus loosened, and soon its anterior projection could be grasped by bone forceps and slowly pulled forward. Several mucoceles were encountered during this dissection. Finally, it was possible to tilt the osteoma out of its cavity. It was then seen that the tumor had been in contact with the roof of the orbit in one place in which there were several small areas of erosion on an otherwise smooth surface. As soon as the tumor had been removed, the eyeball receded. The lateral wall of the left side of the nose was pierced, and drainage was effected by a tube passing from the incision through the cavity of the tumor and out of the left nostril. The skin incision was then completely closed.

The tumor measured 4 by 3.4 by 2.5 cm. It was composed for the most part of dense yellow ivory bone, but in places it was slightly spongy, and an area in the center consisted of spongy bone and pink marrow.

Twelve weeks after the operation there was slight weakness of upward and outward movements of the eyeball with diplopia, slight enophthalmos and a convergent squint.

W. ZENTMAYER.

NASO-ORBITAL RELATIONS DURING INFANCY. ALFREDO ALCAINO, Arch. de oftal. de Buenos Aires **6:** 55 (Jan.-Feb.) 1931.

This is a detailed anatomic study of the relations between the orbital and nasal cavities in the adult and in the child. At birth the ethmoid

contains at least from 2 to 3 cells. During the first year of life it attains a height of 0.5 cm, and a breadth of from 2 to 4 cm., and contains from 3 to 5 cells which are well differentiated. The ethmoid acquires its complete development between the sixth and seventh years; at this age the frontal and ethmoidal sinuses commence to be differentiated from the ethmoid cells, the frontal sinus being well differentiated between the seventh and tenth year and the sphenoid sinus about the twelfth year. The maxillary sinus acquires its definite form by the sixth year (orbital complications of this sinus being exceptional). The author believes that orbito-ocular complications of sinus origin in children under 7 years proceed from the ethmoid.

C. E. FINLAY.

Physiology

THE LIGHT SENSE AS TESTED BY THE PHOTOMETRIC GLASSES OF TSCHERNING. J. H. DELANEY, Am. J. Ophth. **13**: 1058 (Dec.) 1930.

The author remarks that no practical clinical procedure has been adopted to test the light sense. He describes the Tscherning glasses and the manner in which he used them in order to determine their practicability in the office. He comes to the following conclusions:

"1. For a practical test of the light minimum by the ophthalmologist, and for the detection of defects which would be an economic handicap in industry, Tscherning's glasses are an efficient and reliable means of detecting variations. 2. Variations will be met in values obtained from the same individual when tested from day to day. These variations are slight, covering about the same range in value as the variations caused by differences in the size of the pupils. 3. Pre-exposure is not of importance, since, at the end of a half-hour, the patient will have reached the same state of dark adaptation regardless of the pre-exposure. 4. Marked reduction in the light minimum is noted in retinitis pigmentosa and retinitis punctata albescens. 5. Slight changes are noted in the light minimum in such disorders as optic atrophy, retrobulbar neuritis, and neuroretinitis. 6. Tscherning's glasses will detect changes in the light minimum in more advanced cases of glaucoma, but these findings are of less value than a good perimetric examination and visual acuity test. 7. Tscherning's glasses are of no value as an early aid in the diagnosis of glaucoma before other signs are present in the eye."

W. S. REESE.

THE INFLUENCE OF SYMPATHECTOMY ON THE BLOOD-AQUEOUS RELATION. A. LINKSZ, Klin. Wchnschr. **10**: 839 (May 2) 1931.

This paper is a short outline of the author's work on this subject which is to be published in detail in the *Archiv für Augenheilkunde*. Much of late has been contributed by Karczag and Zilahy, Muller and Pflimlin, and Wesselkin on this subject, but they have simply confirmed previous knowledge. Wessely's work during the years from 1900 to 1913 is recalled.

The author concludes that by the blocking or interfering with the permeability of the blood vessels produced by sympathectomy the blood-aqueous relation is changed. For a detailed description of the author's work, the reader is referred to the May, 1931, issue of the *Archiv für Augenheilkunde.*

L. L. MAYER.

IS THERE A CLINICAL REACTION OF THE INTRA-OCULAR PRESSURE OF ONE EYE WHEN THE OTHER IS TRAUMATIZED? SVEN LARSSON, Acta ophth. **8:** 261, 1930.

Experimentally, Weeker demonstrated that a variation in the intra-ocular tension in one eye is accompanied by a consensual reaction in the other. Leplat noted that the traumatized eye shows increased tension and is followed by a phase of hypotension, and frequently the opposite eye shows a similar reaction. Morax and Magitot demonstrated experimentally a hypotony in the one eye when compression is produced in the other. Other investigators showed an increase of protein or fibrin in the untouched eye, but Wessely could not corroborate this finding when he used fluorescein. The author corroborated these findings and noted that an injection of physiologic solution of sodium chloride into the vitreous produces the same change in intra-ocular pressure.

Frequently it is difficult to determine the change in the pressure in the opposite organ because of the gross method employed, the uncertainty of the so-called normal pressure and the normal pressure of the eye before the accident. One may often observe in the clinical picture that there is some variation of the pressure in the undamaged eye. Larsson believes that the change in the pressure cannot be considered as a consensual pressure reaction because the hypotony in the undamaged eye is, as a rule, independent of the pressure in the damaged eye. He concludes that the process must be produced by a vascular reflex action.

A. M. YUDKIN.

Refraction and Accommodation

SURGICAL TREATMENT FOR MYOPIA. ANTONIO J. MANES, Arch. de oftal. de Buenos Aires **6:** 23 (Jan.-Feb.) 1931.

This is a report of nine cases of myopia. In the majority an intracapsular extraction was performed. In a few cases in which the capsule ruptured, the operation was converted into an extracapsular extraction. The results were uniformly good.

C. E. FINLAY.

SUBNORMAL ACCOMMODATION FOLLOWING IRIDOCYCLITIS. N. BLATT, Arch. f. Ophth. **125:** 125, 1930.

Among one hundred and eight cases of iridocyclitis peracta the author found seventeen in which weakness of the accommodation remained as a permanent sequel. This weakness caused either reduced accommodative power or inability to maintain a certain degree of accommodation. The latter was most likely due to a deficiency in the reserve of the accommodative power. The cause and the degree of the disturbance depended on the severity and the duration of the previous iridocyclitis or, in other

words, on the intensity of the pathologic changes in the ciliary body. The general health and the original disease that caused the iridocyclitis had some influence also. The treatment consisted of the wearing of proper glasses, instillations of pilocarpine, application of the faradic current to the ciliary body and exercise. P. C. KRONFELD.

Retina and Optic Nerve

PIGMENTARY DEGENERATION OF THE RETINA IN CEREBROSPINAL SYPHILIS. J. S. FRIEDENWALD, Am. J. Ophth. **13**: 943 (Nov.) 1930.

The author refers to Förster's classic description of syphilitic chorioretinitis of the pigmentosa type in acquired syphilis. He reports three cases of tabes of this type and gives the following summary.

These cases are of interest in that they present a type of optic atrophy associated with tabes that is favorably influenced by treatment. The association of the ophthalmoscopic lesion, which is usually characteristic of congenital syphilis, with tabes and dementia paralytica in acquired syphilis indicates that the nature of the bodily reactions (allergy and immunity) to the spirochete in late neurosyphilis resembles that found in congenital syphilis. Finally, it is of special interest to note that under treatment the amount of retinal pigmentation in these cases showed a decided increase. One must conclude from this fact that once the invasion of pigment into the retina has been stopped, then the normal processes of transport and repair tend to remove the pigment that has already been laid down in the retina. W. S. REESE.

NEPHRITIC RETINITIS: ITS RELATION TO INTRACRANIAL HYPERTENSION. HENRI SCHAEFFER, Presse méd. **39**: 51 (Jan. 10) 1931.

Two interesting cases are reported. In the first case, a young girl presented a picture of albuminuric retinitis, intracranial hypertension and uremia, without retention of chlorides. When first seen, the disks showed edema without hemorrhages or other lesions of the fundi. As the general condition grew more severe, small hemorrhages were noted in the retinas. The vessels showed no congestion. After injections of mercuric cyanide the edema of the disk and hemorrhages of the retina disappeared and the spinal fluid tension was decreased. The author stresses the fact that increased intracranial pressure in such a case must be recognized as a separate pathologic condition from the retinitis. The other case was a chronic nephritic condition with uremia. A typical star figure was noted in the macula, but there were no recent hemorrhages. Treatment with mercuric cyanide was of no avail. In spite of the great increase in spinal fluid pressure there was no edema of the disks or appreciable retention of chlorides.

Thus certain cases of nephritis with increased intracranial pressure show papilledema while others do not. The author believes that nephritis with retention of chlorides does not show a retinitis. On the other hand, the edema of the disk is not due to a retention of water in the tissues. Changes in the blood serum due to albumin, sugar, chlorides or urea seem to regulate the equilibrium of the cerebrospinal fluid.

L. L. MAYER.

A Case of Pseudodetachment of the Retina. H. Lagrange, Arch. de oftal. hispano-am. **31:** 129 (March) 1931.

The case was at first diagnosed by the author and several authorities, including a Lausanne expert, as a detachment of the retina; it subsequently proved to be a proliferating retinitis of undetermined origin. The author places great importance on the employment of a luminous object in the perimetric examination of these doubtful cases.

C. E. Finlay.

Personal Experiences with Gonin's Operation for Detached Retina. J. Igersheimer, Klin. Monatsbl. f. Augenh. **86:** 101 (Jan.) 1931.

The author reports on twenty-one cases of Gonin's ignipuncture, done during the last eighteen months. He considers this operation a decided step forward, but feels the need to define more clearly its limits and possibilities. Dividing his cases into two groups, his first group comprises those that appear unfavorable from the start, either because the detachment is of too long standing, or because previous operative attempts had been made, or on account of a preceding injury. There was partial response in only one of these six cases, that of a boy, aged 15, with a very small hole in a total detachment of three years' standing. The retina became reattached, but the vision remained unchanged due, probably, to the long duration of the detachment.

The second group of fifteen cases presented detachments of only a few weeks' or months' standing. Ignipuncture resulted in reattachment in nine of these cases, although in three of them there was a recurrence within two to three weeks, offering, however, a favorable prognosis. The rupture of the retina had not been precisely reached by the cautery. Of the other cases of this group, two showed deteriorations and six improvement for a period varying from three to seventeen months prior to the publication of the paper. Only slight benefits were obtained in three other patients, two of them with high myopia in their second eyes, and one with a detachment in the eye on which operation was not performed. The unfavorable results in these last three patients were due, in the author's opinion, to thinness of the retina. This possibility should be considered in patients with one eye, on whom Igersheimer ignipunctures only when the detachment is progressing, and if the rupture can be well localized. In absence of a visible rupture, coupled with diminishing vision, he punctures, expecting to find the rupture more readily after temporary reattachment has been obtained. Changes, or hemorrhages or folds in the macula lutea are occasional complications. He observed hemorrhages in the vitreous in two of his twenty-one cases. Perfect reattachment had been obtained in one of these; however, remnants of fibrin were seen in the vitreous a year later. This case also showed increased intra-ocular tension which yielded after the use of miotics for a week. In two other cases, iritis developed, lasting for from three to four weeks after the operation. Incompletely closed ruptures, or tears developing after ignipuncture, will not necessarily produce fresh detachments.

K. L. Stoll.

HETEROTOPIA OF THE MACULA DUE TO CICATRICIAL DISTORTION OF THE RETINA AFTER IGNIPUNCTURE IN DETACHMENT OF THE RETINA: REPORT OF TWO CASES. R. STEIN, Klin. Monatsbl. f. Augenh. **86:** 188 (Feb.) 1931.

Two cases of vertical heterotopia of the macula are reported. It resulted in highly myopic eyes after Gonin's operation for detachment of the retina. The first patient, a woman, aged 30, suffered a detachment after a fist-blow on her eye, a year prior to admission to the hospital. Although very myopic, she had never worn glasses. The vision was reduced to counting fingers at a distance of 40 cm. The field of vision was greatly contracted by a detachment of the upper portion of the retina. A triangular hole was found above the disk. Ignipuncture, done 20 mm. behind the limbus, was followed by a hemorrhage into the vitreous a few days later. Two months after the operation, vision was 1/24 with a 10 diopter concave lens. The patient, however, complained of double vision at a distance, but not at close range when the right eye on which operation was performed was suppressed. The image of the right eye was 20 cm. higher and 25 cm. to the left of the left eye at a distance of 1 meter, while the vertical distance of the double images increased when the patient was looking to her left side. When using the better left eye, the right eye deviated 5 degrees up and 15 degrees out, while fixation of the same object with the right eye caused it to deviate 14 degrees up and 5 degrees out. The ophthalmoscope furnished the explanation. Fine folds were seen leading from the disk downward and temporal. The retinal vessels, leading at first downward and temporal, turned sharply in a horizontal direction, as they left the disk, and crossed the original site of the fovea. The superior vessels lead straight upward into the scar at the site of the cauterization. The fovea itself could be seen 1.5 papilla diameters above and 0.75 of a papilla diameter temporal from the disk, i.e., it had shifted 0.5 papilla diameter toward the nose and 2 upward. The blind spot of the right eye was located about 8 to 9 degrees higher than that of the left eye. Parallel position of the visual axes in a horizontal direction was accomplished by tenotomy of the superior oblique. The heterotopia of the macula was caused by the pull of the shrinking of the scar of cauterization. The author explains the cause of the strabismus, which did not exist prior to the operation, by cicatricial adhesions limiting the function of the superior oblique. A similar case of vertical heterotopia of the macula was reported by Bielschowsky (*Klin. Monatsbl. f. Augenh.* **84:** 755, 1930; abstr. in ARCH. OPHTH. **4:** 942 [Dec.] 1930).

The second case, that of a woman, aged 45, with a myopia of 23 diopters in her right eye, had double vision after intracapsular extraction of the lens. After the operation the picture of this eye was lower than that of the left eye. She had never before complained of diplopia. The heterotopia, in the author's opinion, had been compensated by an abnormal position of the lens.

K. L. STOLL.

DETACHED RETINA IN ECLAMPSIA. E. KLAFTEN, Med. Klin. **27:** 580 (April 17) 1931.

The interesting report by Ingolf Schiötz, which appeared in 1921, is reviewed. The outstanding characteristic was the rapid return of the

retina to normal as the eclampsia was retarded. If one takes Pal's point of view, that an angiospasm is the origin, it is easy to understand why the condition retracts so quickly. Clapps' patient was restored to normal one day post partum, Lotz's two days, and one of Knapes' patients after six days. In four of Schiötz's cases, a normal retina was found from four to six weeks post partum. Since 1885, when von Graefe reported the first case of retinitis in pregnancy, there have been about sixty cases reported in the literature. During the past ten years, in the examination of 25,000 patients during confinement, 206 of whom had eclampsia, showing often retinitis albuminuria, neuroretinitis, hemorrhagic retinitis, papillitis and edema of the retina, only one case of detachment was observed by the author. This case is reported in detail. The outstanding features were the high blood pressure, kidney disease with very marked albuminuria, and, last but most important, the rapid recovery of the retina following therapeutic abortion. The prevention of uremia together with preservation of vision are the indications for early termination of the pregnancy in such cases.

L. L. MAYER.

How Does Detachment of the Retina Occur, and What Are the Prospects of Healing with Proper Care? K. Lindner, Wien. klin. Wchnschr. **44:** 488 (April 10) 1931.

The subject of detachment of the retina is pertinent because of the recent enthusiasm concerning the Gonin operation. Cases due to tumor of the choroid, retinal cysticercus and severe nephritis are not included in the type under discussion. A short review of the history of detachment is given, and some views are expressed as to the method of pathogenesis, with emphasis on Leber's views followed by those of Gonin and Vogt. The fact that the hole in the retina is the initial and most important cause is greatly stressed. Factors that make for the greatest detachment at the onset are brought forth. Normal eyes with detachment do not offer as good an operative prognosis as myopic eyes with degenerated vitreous. The prognosis is very much poorer in detachments existing a year and a half or longer. Experimentation on animals' eyes has shown that the cautery causes adhesion between the retina and choroid. Improvement of the Gonin method is left for the future. A footnote refers to the new Guist operation (November, 1929) with potassium hydroxide as the cauterizing material, which has given the best results.

L. L. MAYER.

The Relation Between Retinitis Pigmentosa and Glaucoma. S. Z. Kotliarevskaya, Russk. arch. opht. **8:** 159 (May) 1931.

The author reported two cases of retinitis pigmentosa. One of these patients suffered from chronic simple glaucoma in one eye. The second patient lost his right eye because of inflammatory glaucoma; he had poor vision in the left eye with glaucomatous excavation of the disk and nasal contraction of the visual fields.

Thirty-one cases (including these two) of retinitis pigmentosa combined with glaucoma have been reported in the literature. The few theories explaining the rare occurrence of these two diseases together

are based on the presumption that glaucoma can occur in retinitis pigmentosa as a result of development of connective tissue between the choroid and the retina and also because of sclerosis and obliteration of the vessels (i. e., the basic cause is disease of the vascular system). None of these theories, however, explains sufficiently why the combination of these two diseases is so rare.

O. SITCHEVSKA.

Trachoma

A DISCUSSION OF THE ETIOLOGY OF TRACHOMA WITH SPECIAL REFERENCE TO BACTERIUM GRANULOSIS (NOGUCHI). R. P. WILSON, Brit. J. Ophth. **15**: 433 (Aug.) 1931.

After some general remarks on past and present views on the etiology of trachoma, the author summarizes the present position with regard to "cell inclusions" and filtrable viruses. He then considers the recent researches of Noguchi on the etiology of trachoma and notes especially the work that has been done since Noguchi's original monograph was published. He reviews the work done in conjunction with Thygeson and Abassi: Neither of the strains of suspicious gram-negative bacilli, which Thygeson had isolated by himself, gave positive results in any of the monkeys, even after three heavy inoculations on successive days. With small pieces of infected conjunctiva from monkeys brought to Egypt by Thygeson, positive results were obtained. The uninoculated eye was still healthy on the thirty-eighth day. This eye was now inoculated with secretion from the fellow eye, and a positive result was obtained. From this monkey, a positive result was obtained in a Macacus rhesus. As a result of caging this monkey with others, it was demonstrated that infection may occur as a result of cohabitation.

Macacus rhesus inoculated with human trachomatous material developed a condition absolutely indistinguishable from a mild case of spontaneous folliculosis.

Experimentation on the transmission of both virulent strains supplied by Thygeson to the conjunctiva of a man with mild catarrhal conjunctivitis resulted in an inflammation of the lids, granular in appearance with a slight pericorneal congestion; the condition lasted about one week, leaving no sequelae. Monkey tissue transfer gave negative results.

The lesions, which are induced by tissue transfers from one infected monkey to another, in certain respects resemble the human disease, but the tarsus is seldom involved, pannus has never been observed, the conjunctiva although thickened and raised by subepithelial follicles always remains smooth and glistening, and the lesions are very localized and have only rarely led to definite scar formation; indeed, the clinical picture is just one of a severe folliculosis. It is also of interest to note here Lindner's opinion on the monkeys that he saw at the Rockefeller Institute. He says: "There is no doubt in my mind whatsoever that Noguchi's experimentally produced conditions have nothing in common with trachoma. It can scarcely be doubted that Noguchi has discovered not the cause of trachoma but one of the causes of folliculosis of the conjunctiva.

W. ZENTMAYER.

Therapeutics

ULTRAVIOLET LAMP FOR FOCAL TREATMENT OF DISEASES OF THE EYE. A. GUTMANN, Klin. Monatsbl. f. Augenh. 85: 782 (Dec.) 1930.

The author has constructed an improved model of his ultraviolet lamp for focal treatment of diseases of the eye which he describes in detail. The intensity of the ultraviolet rays is reduced by the insertion of a diaphragm 12.50 mm. in diameter. This will produce an erythema dose of 6,000 volt-seconds when a lens of 4 mm. is used for three minutes and forty-five seconds under focal illumination. The best results, however, are obtained with an average intensity of exposure with a 40 mm. lens for one and a half minutes per dose. The lids, their margins and the conjunctiva are treated with only from 30 to 50 per cent of the skin erythema dose. This dose is essential to prevent edema of the subcutaneous tissue with secondary pigmentation, conjunctivitis and keratitis. Eczema of the lids is treated by allowing the rays to move over the diseased area for two minutes, at the most, at each treatment while the eyes are closed. Either the lamp, which is placed on a glass plate, or the patient's head may be moved for this purpose. Simple and ulcerative eczema of the palpebral margin, coupled with secondary loss of the cilia, are treated after touching the margin with a 2 per cent solution of fluoresceine-sodium, or fluoresceine-potassium. The margins of the lids are slightly everted during the treatment. Four to five treatments will check itching and burning of the lids in chronic conjunctivitis, after the usual therapy has failed; the lids are everted and their inner surfaces and the fornices are treated by moving the light over each lid for one and a half minutes. The treatment is repeated on the following four to ten days, with intermissions of a day, if required. Recurrences of eczematous and scrofulous conjunctivitis have yielded good results in connection with the usual treatment. Scrofulous keratitis is treated by concentration of the focus on each infiltration from one and a half to two minutes steadily. From five to ten treatments are usually sufficient to bring about a cure, even in unruly children, in whom the focus is allowed to move over the diseased area. The lids are kept apart. Recurrences have been observed after treatment, and injections of tuberculin, albumin, diet and irradiation of the skin of the body have been resorted to. Serpent ulcers of the cornea are treated for two minutes twice daily by means of the customary therapy. Hypopyon disappeared promptly and fainter scars with better vision than those resulting after other forms of treatment were recorded. Cauterization should be avoided. Gutmann's apparatus, containing some rays up to 500 microns and causing no blinding effects, sends the rays into the deeper layers of the cornea and to Descemet's membrane. Parenchymatous keratitis, therefore, can be treated with good results, although the ultraviolet rays will not penetrate to such a depth. Iritis, coupled with serpent ulcers, can be cured for the reason stated, and posterior synechiae can be prevented. Sixty per cent of cases of herpes of the cornea were cured in from eight to ten days, leaving no extensive or dense scars. Various kinds of iritis were cured after the insertion of a second filter of uviol glass or by replacing the quartz lens with a lens of 20 diopters of the trial case. This means allows the luminous rays to reach the eye without causing photophobia. Tuber-

cles, papules and gummas of the iris are treated by focusing the rays directly on them for two minutes. Damage to the iris was never observed after these treatments, which helped to shorten the process of recovery. The rules of individual general treatments have to be observed.

K. L. STOLL.

AMBLYOPIA CAUSED BY THE ABUSE OF ALCOHOL AND TOBACCO; TREATMENT WITH COMPOUND SODIUM NITRITE SOLUTION. R. PFLIMLIN, Klin. Monatsbl. f. Augenh. **85**: 787 (Dec.) 1930.

A compound sodium nitrite solution is given the preference to other vasculodilating preparations at the eye clinic of the University of Basel in the treatment for amblyopia caused jointly by alcohol and tobacco. It consists of a 2 to 4 per cent solution of sodium nitrite, to which physiologic solution of sodium chloride and alkaline phosphates are added. The lowest lethal dose for rabbits is from 0.06 to 0.08 Gm. per kilogram of body weight. It relaxes the walls of the capillaries and arterioles. No direct action on the heart and no central or peripheral paralyses of the nervous system were noticed. The drug is used to reduce the blood pressure, and prior to operations requiring the opening of the globe.

Patients with tobacco-alcohol amblyopia were treated with intramuscular injections of 1 cc. of the compound sodium nitrite solution every second or third day, beginning with a concentration of 0.02 and applying a concentration of 0.04 after four or five injections. In some cases results were obtained after from eight to ten injections; in others, after twenty. Treatments were discontinued early in a number of patients, when congestion, vertigo, malaise or an acute rise of the temperature occurred. The fundus, examined with red-free light, showed a marbleization of the macular region, diminution of the yellow, distortion or total absence of the reflex of the fovea and an incomplete or entirely absent design in the nerve fibers. These symptoms remained unchanged in all but one case after vision had become normal again; they could still be observed after from one to two years. Pallor of the entire disk or its temporal portion had changed to a pink hue in some cases; this is explained as the result of the elimination of the spasm of the vessels. A table records the condition of the disk and macula at the time of the patient's admission to and discharge from the hospital, and also the number of the injections and their results. Abstinence from tobacco and alcohol will not suffice to bring about a cure in every case. The results are decidedly more prompt after the use of the compound solution of sodium nitrite. Changes in intraocular tension were not observed. The age of the patients ranged between 50 and 60 years, and all of them had arteriosclerosis. Their blood pressure was 180 mm. or more. It decreased during the treatment, but began to rise again after from three to five months. Pflimlin suspects some connection between this toxic amblyopia and arteriosclerosis. The treatments were continued under ambulatory conditions after a certain number of injections had been well stood by the patient. Special care will have to be given to persons suffering from arteriosclerosis of the brain.

K. L. STOLL.

IMRE'S TREATMENT OF DISEASES OF THE FUNDUS WITH AMYL NITRITE. MARIE PROCKSCH, Klin. Monatsbl. f. Augenh. **85:** 795 (Dec.) 1930.

Satisfactory results were obtained by inhalation of amyl nitrite in a number of patients with retinitis pigmentosa. The results were negligible in atrophy of the optic nerve; they varied in three cases with atrophy of the choroid; three other patients were slightly improved, and three decidedly improved. The treatment consists in inhalations of one or two drops of amyl nitrite on four or five consecutive days, when the maximal action is usually reached. Improvement of the light sense has been observed in persons with normal and with diseased eyes.

As measured by the Birch-Hirschfeld adaptometer, amyl nitrite furnishes results by improving adaptation but not by regeneration of real atrophic tissues. Its use is especially recommended in cases of retinitis pigmentosa, in high myopia and in various kinds of chorioretinal atrophies.

K. L. STOLL.

ON THE INDICATIONS FOR SPECIFIC THERAPY FOR TUBERCULOSIS OF THE EYE. A. Y. SAMOILOFF and V. P. HOCHBAN, Russk. arch. opht. **8:** 16 (May) 1931.

The authors believe that in all cases of tuberculosis of the eye, a careful general examination with a roentgenologic picture of the lungs should be made before treatment with tuberculin is begun.

They report one case of tuberculous keratitis and one case of choroiditis that were combined with active tuberculosis of the lungs. One patient suffered from a sluggish tuberculous keratitis with occasional exacerbation; the x-ray picture showed an arrested process of the lungs. Tuberculin treatment in that case activated the old lesion of the lungs without provoking a focal reaction. In the fourth case, with an old tuberculous lesion of the cornea in which the Mantoux test was positive, neither the intradermal nor the subcutaneous injections of tuberculin gave a focal or general reaction. In some cases of Ranke II (period of hypersensitivity) cautious tuberculin therapy in very small doses may give good results. In tuberculous choroiditis, and in iritis with tubercles, intradermal injections should be given until desensitization is obtained.

The authors divide all cases of tuberculosis of the eye, in regard to indications for tuberculin therapy, into the following groups: 1. Specific therapy in various tuberculous affections of the eye with active tuberculosis of the lungs is absolutely contraindicated. 2. In cases of sluggish tuberculous keratitis and kerato-iritis in which a general reaction is produced before the focal reaction, treatment with tuberculin should not be used. 3. In cases of iritis with tubercles, the patients can be given subcutaneous injections only after desensitization is obtained by means of intradermal injections. 4. All other patients with tuberculosis of the eye should be subjected to specific therapy, the initial dose not exceeding 0.2 cc. of a 1:1,000,000 dilution.

O. SITCHEVSKA.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

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WILLIAM C. FINNOFF, M.D., *Secretary*

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GEORGE F. SUKER, M.D., *Chairman*

ADULT HEREDITARY ANTERIOR MEGALOPHTHALMUS SINE GLAUCOMA: A DEFINITE DISEASE ENTITY, WITH SPECIAL REFERENCE TO THE EXTRACTION OF CATARACT. DR. DERRICK T. VAIL, JR., Cincinnati.

This article with the discussion appeared in full in the ARCHIVES OF OPHTHALMOLOGY for July, 1931, p. 39.

HEREDITARY JUVENILE GLAUCOMA SIMPLEX. DR. ROBERT H. COURTNEY and DR. EMORY HILL, Richmond, Va.

An attempt is made to bring the literature on this subject down to date and review some of the previously reported families. A report is made of a family in which the disease has been present for five generations. Case histories of the more affected members are given.

It seems clear, both from a review of the literature and from a study of the family reported, that there is a definite type of glaucoma which is hereditary. This type of glaucoma occurs usually in the second or third decade of life, and is not accompanied by the recognized conditions occurring in glaucoma of later life, i. e., a small cornea, a shallow anterior chamber, ametropia and a large lens, nor is it associated with other congenital defects, such as aniridia and colobomas.

It is probable that the glaucoma factor is dominant over the factor for normal eyes and that it follows the same hereditary laws as brown irides.

While the hereditary type of juvenile glaucoma logically falls into the classification of chronic glaucoma to distinguish it from the acute congestive type, it must be recognized that the disease in reality runs a rather rapid course, and that early diagnosis and early surgical intervention are essential.

There is no definite evidence that the disease can be influenced by miotics or other hypotensive drugs.

(This article will appear in full in THE JOURNAL of the American Medical Association.)

DISCUSSION

DR. EDWARD SHUMWAY, Philadelphia: That glaucoma occurs at an early age, under 30 years, in an inherited form is now a well established fact. One of the first to report a long series of cases was a Philadelphian, Dr. George C. Harlan, who, in 1885, published an example under the

heading, "Glaucoma Appearing in Five Successive Generations Before the Age of 20." Later papers, particularly those by Lawford and Nettleship in England, and by Löhlein and Plocher in Germany, have established the condition as a clinical entity, and the literature on the subject has been summarized in Peter's article in the third edition of the Graefe-Saemisch Handbuch. Few instances have been recorded in which more than three generations were followed, and I desire to place on record the history of a family in which juvenile glaucoma occurred in four successive generations. (For a full report of this family see *Transactions of the Section on Ophthalmology of the American Medical Association,* 1931.)

The family shows a record of juvenile glaucoma in members of four generations, all of whom developed the disease before the sixteenth year and, with the exception of the last patient who is 16 now, all gradually lost their sight, with or without operation. Of the six persons, five were females and one a male, two males in the second generation escaping. I have little to add in the general discussion, as the authors of the paper have given a good résumé of the facts as they have been reported in the literature. Usually the hereditary form of juvenile glaucoma is chronic in type; it shows, moreover, the peculiar phenomenon of "anticipation," that is, the appearance in ever lessening age in the succeeding generations, and a long lasting prodromal stage is noticeable. It has been generally agreed that the patients become nearsighted because of the glaucoma, and not vice versa. There is no preference for either sex, as a rule, although in my series the females largely predominated. From a prognostic and therapeutic point of view juvenile glaucoma shows a decided malignity. Miotics and various forms of operation have had little permanent effect, but the consensus in later years seems to be that the Elliot trephine operation offers the most hope of success, if vision progressively fails.

Dr. John O. McReynolds, Dallas, Texas: It has been my observation that the form of the condition observed in our country is not a frequent one. Those cases developing in the second and third decade of life have been exceedingly infrequent. They actually occur, but the most frequent form that we have observed which could be properly called hereditary glaucoma has developed in the first decade of life and has participated somewhat in the characteristics of buphthalmos rather than the type described. Of course I feel that it is a condition depending essentially on anatomic changes in the eye, and because the other depends on various causes, as brought out so well by Drs. Magitot and Duke-Elder and others, we are impressed with the fact that we have not come to a full explanation of many of these cases, other than that they depend on anatomic changes in the eyeball and on differences in the nervous system.

Dr. J. W. Jervey, Greenville, S. C.: From clinical observation I have come to believe that there is a hereditary tendency in all forms of glaucoma. If the present-day conception of the physiology of intraocular drainage is correct, no ordinary iridectomy need be expected to reduce tension satisfactorily (save by the happy chance of an unexpectedly filtrating wound) in an eye with a deep anterior chamber and thereby a presumptively open canal of Schlemm. In such an eye addi-

tional and complementary drainage must be obtained by means of one of the fistulizing operations.

DR. M. GOLDENBURG, Chicago: It is my belief that in addition to the inherited anatomic factors brought out by the authors, there is an inherited tissue sensitivity, as demonstrated in the increased capillary permeability, which may be secondary to, or preceded by, an alteration in the chemical composition of the body fluids. These two factors are difficult to separate, and it is impossible to state which is the more important or which takes precedence in the precipitation of symptoms.

DR. F. H. VERHOEFF, Boston: In two patients with juvenile glaucoma examined with the microscope, I found changes in the filtration angle that were similar to the changes found in buphthalmos. These observations make me believe that these two conditions are essentially the same, differing only in degree. Both of them depend, ordinarily, on an imperfect filtration angle. In other words, juvenile glaucoma is that form in which the filtration is not completely cut off, and in which, therefore, in early life there is not enough pressure to injure the globe, whereas in buphthalmos the pressure is sufficient. There are different kinds of buphthalmos. There may be no symptoms in one eye and loss of elasticity in the other. So it seems to me that juvenile glaucoma and buphthalmos simply represent differences in degree of the same condition.

DR. EMORY HILL: The history of the hereditary type of juvenile glaucoma logically falls into the classification of chronic glaucoma and the very acute type. This runs a rather rapid course. Early surgical intervention we think should be done. I do not favor medical treatment. We have found no record in the literature of miotics having done any good. I think that Dr. Jervey's point is well taken that something more than a simple iridectomy should be done in these cases.

THE NATURE AND ORIGIN OF THE PIGMENTED STREAKS CAUSED BY SEPARATION OF THE CHOROID. DR. F. H. VERHOEFF, Boston.

From macroscopic and microscopic studies of two eyes showing pigmented streaks in the fundus, and of five eyes in which separation of the choroid still existed, the following conclusions are drawn:

Pigmented streaks in the fundus resulting from separation of the choroid are entirely different in nature and origin from angioid streaks.

Very fine pigmented streaks exist while the choroid is still separated. These are furrow-like creases in the surface of the choroid. They are not usually visible on ordinary ophthalmoscopic examination, but they may be seen, in some cases at least, by transillumination of the separated choroid through the sclera. All these creases disappear when the separation subsides.

The permanent pigmented streaks, seen after the separation has subsided, are ridgelike thickenings of the pigment epithelium. The pigment cells composing the permanent streaks originally accumulate within some of the furrow-like creases that exist while the choroid is separated. When the choroid becomes reapplied to the sclera and these creases are smoothed out, the accumulations of pigment cells are elevated, thus giving the appearance of streaks.

(This article will appear in full in THE JOURNAL of the American Medical Association.)

DISCUSSION

DR. T. B. HOLLOWAY, Philadelphia: Dr. Verhoeff has been unfortunate in not having seen the pigmentation in these cases with the ophthalmoscope, but just as I am of the opinion that subsequent pathologic studies will verify his observations concerning angioid streaks, just so I believe that his explanations and conclusions concerning the present type of pigmentation will be accepted. They are satisfying as far as my personal opinion is concerned.

DR. E. V. L. BROWN, Chicago: One characteristic of the detachment streaks is not sufficiently stressed or discussed by Dr. Verhoeff, although it is definitely present and described in each of his cases, namely, their predominantly longitudinal and circular course. It seems to have been present in practically all other reported cases.

E. Fuchs has stated that the folds are of two kinds, meridional and circular, and that the posterior folds represent a telescoping of the detached choroid over the firmly attached portion around the disk. This posterior telescoping is well shown in Plocher's drawing of detachment (Klin. Monatsbl. f. Augenh. **59:** 610, 1917). In this there is an obvious reduction of surface expanse of the choroid from before backward. A reduction of equatorial (circumferential) expanse occurs when the separated choroid bulges inward toward the axis of the eye and the longitudinal folds or creases of the choroid are thus produced. Anteriorly, the same concentric folding occurs from bending or overlapping of the preequatorial choroid onto the most anterior choroid. Meridional folding comes about the more readily because it coincides with the course of direction of most of the larger vessels of the choroid. Oblique streaks do occur.

I wish to call attention to a third kind which apparently is rare, or at least concerning which there has been very little written, namely, the form due to arteriosclerosis of the choroidal vessels. This was first described clinically in two cases (and anatomically in one of the cases) by Siegrist in 1899.

DR. F. H. VERHOEFF: I am aware, of course, that pigmented streaks sometimes result from separation of the retina, and I have seen them in cases of high myopia and other conditions, but such streaks occur so rarely and are so scanty when they do occur that they do not properly come within the scope of my paper. With the streaks described by Siegrist, however, I was unacquainted until his paper and that of Koeppe were called to my attention by Dr. Brown a few days ago.

These streaks have no relation to postoperative separation of the choroid, but they are pertinent to my paper because they are histologically similar to the streaks investigated by me. Siegrist could offer no satisfactory explanation for their occurrence. Three possible explanations in addition to those offered by him suggest themselves to me: first, that they are due to antecedent, spontaneous separation of the choroid; second, that they result from previous creases in the choroid, and third, that they are caused by the pulsation of certain choroidal vessels. When there is marked sclerosis of the choroidal vessels it is reasonable to assume that for a time some of the latter may pulsate to an unusual degree and that the pulsation may locally stimulate the pigment epithelium to proliferate. This theory seems to me the most probable one.

THE CLINICAL RESULTS OF THE INTRANASAL TEAR SAC OPERATION.
DR. J. M. WEST, Allentown, Pa.

Dr. West described his operation and showed a number of lantern slides to illustrate the technic and the beneficial results of the operation.

DISCUSSION

DR. J. SHELDON CLARK, Freeport, Ill.: Dr. West has always stressed the point of having restored function and a lack of epiphora as end-results of his operation. To my mind this is the crux of the whole proposition. Chemical cauterization and external incision will cure a large percentage of the cases, but there is another complaint—epiphora—which can be a source of great discomfort to the patient, who, after all, is the one most to be considered. Again, epiphora is one of the indications for this operation.

Dr. West operates in the presence of phlegmon. I have never had the courage to do so. Surely all one can do with the external procedures in such cases is to open and drain and await a favorable time for further work.

A good and proved point made by Dr. West is that pneumococci in the conjunctival sac disappear after the intranasal operation, but that they do persist after the external operations—and in 43 per cent of the cases—should set at rest the fears entertained regarding the efficiency of drainage and the complete clearing up of infection after the endonasal operation. This should be of great interest to those who contemplate a cataract extraction subsequent to operation on the sac. This has always been a bugbear in the minds of many oculists.

Variations from the normal may make the endonasal operation impossible without preliminary preparation, such as resection of a deflected septum, resection of overhanging middle turbinals and, in some cases, resection of the tip of the pyriform process—all this work being done as a preliminary step, so as to enable the operator to see the area on which he wishes to work. Such situations, I believe, call for the combined method of operating, in which most of the work is done as heretofore described, extranasally.

In disease of the nasolacrimal system it is well to keep in mind the dictum of Schaeffer, who said: "The success or failure of non-surgical treatment of the diseased nasolacrimal passageways is largely dependent upon the type of nasolacrimal duct and ostium encountered."

DR. W. B. CHAMBERLIN, Cleveland: Personally, I am very enthusiastic about Dr. West's operation, and perform it in preference to all others. I have not performed it in the presence of phlegmon or when the sac has been excised in toto, nor have I performed it on children.

DR. GEORGE S. DERBY, Boston: I was very much surprised and interested in Dr. West's statement that ophthalmologists are still treating patients with dacryostenosis and dacryocystitis by the old methods of irrigation, probing and excision of the sac, so I asked my intern to look up the cases that we have had at the Massachusetts Eye and Ear Infirmary during the past six months. We found that forty-seven operations had been performed on the sac, and of these thirty-seven were dacryocystorhinostomies of one sort or another, and ten were simple excisions of the sac. These figures do not seem to bear out Dr. West's contention,

and I should like to ask him on what he bases his statement. We still find that in certain cases it is wise to excise the sac, as in ulcer of the cornea when we want to get quick results, and in certain old persons on whom we wish to do a cataract extraction without much delay.

DR. J. M. WEST: My idea about the treatment for dacrocystitis is based on what I found when I visited the different clinics in Philadelphia and New York, where I saw nothing but probing of the nasal duct and extirpation of the tear sac.

Dr. Clark stressed what I wanted to emphasize, but did not have time. If one is going to do a cataract operation and finds the streptococcus or pneumococcus on taking a culture, one irrigates the canal and some milky fluid comes through; when the tear sac operation is performed, one will find a clean field for the cataract operation. The ophthalmologist might think that the patient would blow nasal secretions into the conjunctival sac, but that practically never happens. It is the easiest thing in the world to avoid. Take a small piece of gauze and push it up in the nose where one makes the artificial window. The patient cannot possibly contaminate the tear sac by blowing his nose. Mattice found infection in the tear sac a year after extirpation. Cultures were made, and 43 per cent of the cases still showed the pneumococcus. In Berlin we found that the pneumococcus and streptococcus usually disappeared the first or second day after operation.

THE PATHOLOGY AND TREATMENT OF CORNEAL ULCERS. DR. OSCAR B. NUGENT, Chicago.

Dr. Nugent described his technic of applying ultraviolet irradiation from a Birch-Hirschfeld carbon arc lamp in the treatment for corneal ulcers.

(This article will appear in full in THE JOURNAL of the American Medical Association.)

DISCUSSION

DR. LUTHER C. PETER, Philadelphia: It is generally believed that trauma followed by bacterial invasion is the chief etiologic factor in the formation of corneal ulcers. In a small number of cases severe conjunctival infections alone may be sufficient to bring about ulcer formation without evidence of trauma.

In the active treatment of ulcers it is of the greatest importance to know from smears and especially from cultures the bacterial growths that are active in the destructive process. With this knowledge one is fortified and prepared to meet the demands of an individual case intelligently and effectively. For example, in the serpiginous form of ulcer the active micro-organisms may be many, but the pneumococcus, the staphylococcus and the diplobacillus of Morax-Axenfeld are the chief offenders. Axenfeld claimed that he had never seen a serpiginous ulcer due to the diplobacillus that would not yield to zinc preparations. This observation is worthy of serious consideration. It entails a method of treatment that is far less destructive than the radical methods to which we sometimes must resort.

The essayist's method of using the Birch-Hirschfeld carbon arc lamp is to be commended. It is worthy of thorough trial as less destructive than the cautery or thermophore, and yet active in sterilizing a necrotic

area. In addition to this method, I wish to call attention to pasteurization as an efficient means of stimulating normal nutritive processes without the destruction of tissue that is necessarily involved in the use of the cautery and thermophore. On the other hand, there are instances in which only the actual cautery will arrest the process.

DR. JOHN GREEN, St. Louis: The subject of corneal ulcers deserves the earnest consideration of every ophthalmologist. If Evans' estimate that 6.4 per cent of elderly blind people are blind from the sequelae of corneal ulceration is truly representative, then it behooves us to do all in our power to prevent the development of corneal ulcer or to adopt such measures, once it is established, as will promote healing with the minimal amount of cicatricial tissue.

I would modify Dr. Nugent's statement that "most corneal opacities are caused by ulceration" to "most corneal opacities that result in serious impairment of vision are caused by ulceration."

In cataloging some of the recent suggestions in therapy, Dr. Nugent has failed to mention two methods, which, in my hands, have proved of utmost value: 1. Thermotherapy, under known conditions of size of applicator, degree of heat and duration of application. This is now possible with the electric thermophore (Shahan), originally advocated as a treatment for serpiginous ulcer of pneumococcus origin, for which it has proved eminently successful; it has also proved efficacious in the ulcer of dendritic keratitis, in ring ulcer and in the tiny ulcer that sometimes appears at the apex of the cone in keratoconus. 2. Coverage of the ulcerated area with partial or complete conjunctival flaps. My own experience with ultraviolet therapy is more recent and much more limited than Dr. Nugent's, but my results have been extremely favorable.

DR. CHARLES A. BAHN, New Orleans: Several years' experience with ultraviolet therapy in corneal ulcers, including the use of both the Zeiss and the Bausch and Lomb lamp, have convinced me that ultraviolet irradiation is an excellent auxiliary to other treatment, especially in pneumococcal and herpetiform ulcers. I think a word of warning, however, should be sounded, that one should not rely entirely on this means, especially at the critical stage of pneumococcal ulcers, in which several days' delay would change the expected results of treatment and might mean the loss of the eye.

DR. P. C. JAMESON, Brooklyn: It is my own feeling, and I feel sure that of the essayist, that in practicing the new remedies we should not abandon the old ones, many of which he mentions. It is with the old remedies that most of us have fought many a battle, and what sometimes seemed to be a hopeless battle, and won. While we should preserve an open mind in the reception and trial of the new, many well tried old remedies and procedures, the acquisition of many years of cumulative experience, will remain the anchor sheet in the treatment for corneal ulcer.

DR. OSCAR B. NUGENT: I should like to say a word about the methods of applying ultraviolet rays. I have had experience with but one method, that of using the Birch-Hirschfeld lamp. This necessarily yields a weaker ultraviolet ray than is produced by the superheated mercury

vapor filter. Ultraviolet irradiation is not suitable for all corneal ulcers. I use it in all cases in which the ulcer is due to dystrophy, to combat bacterial infection which is likely to occur.

THE NEED OF MEDICAL SOCIAL SERVICE IN EYE CLINICS. DR. GEORGE S. DERBY, Boston.

Social service work in eye clinics deserves more widespread recognition than it receives at present. At the Massachusetts Eye and Ear Infirmary it has increased hospital efficiency by at least 50 per cent. Without social service hospital methods are wasteful, as patients often do not follow the directions given them and do not get the treatment prescribed. In many instances the patient does not report back, and often wanders from clinic to clinic.

A painstaking examination and careful prescription on the part of the doctor is wasted if the patient does not do his part. Through social service much of this waste can be eliminated: (1) by furnishing the personal touch to the case, which the busy doctor is often unable to give; (2) by follow-up work in the home; (3) by making use of the many medical, social and charitable agencies that exist in the community, in this way making treatment available, and (4) by doing intensive work in certain groups of cases.

Social service thus becomes one of the most potent agencies that we possess to combat loss of vision and in the prevention of blindness. Its further development in eye hospitals is greatly to be desired.

(This article will appear in full in THE JOURNAL of the American Medical Association.)

DISCUSSION

DR. J. HERBERT WAITE, Boston: In crowded clinics the doctor must concentrate on the medical problems of the patient, and the personal touch is largely lost. Social service furnishes right-hand aid to the physician by attacking the social, psychologic and economic problems of illness. The day of the individual worker is past, and the day of organized team work is at hand. Of this team the social worker is an important and indispensable member. Those of us who have been fortunate enough to know by experience the worth of social service to an eye clinic would view with alarm the crippling effect of its withdrawal. We would just as soon give up our roentgen departments and our clinical laboratories as permit our medical social workers to depart from our midst. We have come to look on social workers as indispensable aids to our work and real consultants in our problems.

DR. HARVEY J. HOWARD, St. Louis: When one analyzes the records of cases of blindness, one is appalled by the information that almost all of this blindness could have been prevented: that it was caused by somebody's ignorance. I have no sympathy with the attitude "that a man's eyes are his own and that he may bring them to the clinic for cure or keep them at home, as he sees fit." In fact, I think that this whole question of medical social service hinges on our agreement or disagreement with this attitude.

The ways in which a well trained, sympathetic medical social worker can assist the ophthalmologists are too numerous to mention. One of the most important functions is to interpret to the patient in simple terms

the doctor's diagnosis, his directions and the probable prognosis. The vital thing is to get the patient to carry out the treatment prescribed and to come back when directed. The medical social service is the channel for bringing the patient back. It also acts as a steering agent for welfare agencies whose cooperation may be required in the home of that patient before he can come back and before the directions of the doctor can be carried out. The medical social worker is also the liaison officer to connect the eye clinic service with other clinical and hospital services. In the handling of inflammatory diseases of the eye speed is necessary. Prompt action and hospitalization can be best brought about by the medical social worker, but in many cases she will have to visit the patient's home, and secure the cooperation of other welfare agencies before this can be done.

DR. G. E. DE SCHWEINITZ, Philadelphia: I venture to quote some advice of my own recorded in an address delivered sixteen years ago before the National Committee for the Prevention of Blindness. The social worker who makes a calendar of glaucoma patients in any hospital and properly follows them up will do much, for it is through this channel that education most readily passes to the races among which this disease is the most prevalent and which furnish the best customers of the eye clinics. Furthermore, an important function of social service is concerned with the recognition of symptoms in those who sadly need attention, but who have never presented themselves for examination, not only in the house that is visited, but also in the neighbor's house; thus the knowledge of the symptoms of any disease that demands prompt treatment quickly filters through a crowded street. Such a disease is glaucoma, with its long prodromal period, just the period during which the disease can be stopped before it has produced changes that are practically irremediable. To many other ocular conditions the same plan of prevention applies. Therefore, it would seem that a national committee for the prevention of blindness should control a social service as part of its equipment, whose workers should specialize in ocular diseases, but who should in this respect assist in the extension of this work of the hospital physician into the life and home of the patient, and who would be attached to eye clinics and dispensaries which cannot afford the important help that comes from trained service of this character.

DR. F. PARK LEWIS, Buffalo: Five years ago a committee was appointed by the New York Academy of Medicine, of which Dr. Berens was chairman, to make a study of this question. They brought in a most amazing report in which it was found that in various clinics it was not at all uncommon for people with corneal ulcer, glaucoma and various other diseases that threaten the integrity of the eye to appear once, twice or possibly three or four times and then disappear, probably visiting other clinics. They might not reappear for six, eight or ten months; then they would come back with destruction that was beyond repair. I therefore feel that the introduction of this social service is of such great importance that I hope it will be inaugurated in other clinics throughout the country. The National Society for the Prevention of Blindness gives its most hearty and cordial support.

ORBITAL METASTASIS FROM MALIGNANT TUMORS OF THE SUPRARENAL. DR. FRANK E. BURCH, ST. PAUL.

This article with the discussion will appear in full in a later issue of the ARCHIVES OF OPHTHALMOLOGY.

DISCUSSION

DR. PETER C. KRONFELD, Chicago: From Dr. Burch's presentation and from a study of the literature we have learned that metastatic tumors of the orbit are not rare. There is no other tumor that provides us so well with clinical and anatomic material of metastatic lesions as the hypernephromas. The actual frequency of metastatic hypernephomas in the orbit is around 0.25 per cent.

Dr. Burch mentioned convulsive attacks and a hemiparesthesia, perhaps a hemihypesthesia, in the course of the disease. The postmortem report does not contain any statements concerning the observations on and within the skull. The orbital tumor did not extend into the cranial cavity. Therefore I should like to ask Dr. Burch whether the skull was opened and what was found there.

Pathologists are loath to accept bilateral carcinoma of the suprarenals as primary, because it is much more often a metastasis. From Dr. Burch's microscopic sections of the liver and orbit I should have made a diagnosis of metastatic malignant sarcoma, most likely originating from the skeleton or lungs. Were these examined for a primary focus? If there were no other tumors that could be considered as the primary lesions, then we have to accept this case as one of the very rare suprarenal carcinomas which, from the resemblance of their metastases to sarcomas, have been and probably still should be classified as mesotheliomas, following the suggestion of Adami.

DR. PARKER HEATH, Detroit: There are three convenient, though incomplete, clinical groups: (1) the Grawitz neoplasm, a nephrogenic mesothelioma; (2) the true cortical suprarenal type, sometimes with associated constitutional changes, and (3) the true mixed tumor of the infantile kidney, a renal blastoma, causing early death of the patient.

Suprarenal tumors tend to metastasize to the bones of the pelvis, the lungs and, by the laws of chance, the orbit. Frequently study of the metastasized material from the renal blastoma shows cells resembling those of sarcoma, sometimes those of rhabdomyosarcoma.

Secondary constitutional changes follow irritative lesions of the suprarenal cortex as: hirsutism, an increase in muscular power, secondary sexual manifestations, sudden high hypertension, early degeneration of the blood vascular system and polycythemia. These mesodermal reactions as remote, indirect effects of irritative malignant conditions of the suprarenal cortex are of the greatest value in the differential diagnosis and management of the patient.

DR. F. H. VERHOEFF, Boston: The rosettes of neuroblastoma are entirely different in nature from those of retinoblastoma. In the patients with neuroblastoma whom I have examined, the rosettes represent an attempt to form a sympathetic ganglion and show embryonic nerve fibers.

Dr. Frank E. Burch: The cranium was carefully studied, and no evidence to explain the convulsive attack was found. No roentgenogram of the long bones was made.

Chiasmal Symptoms in Intracranial Tumors. Dr. Loyal Davis, Chicago.

This article with the discussion appeared in full in the Archives of Ophthalmology for August, 1931, p. 181.

The Ductless Glands as They Appertain to Eye Diseases and to Surgery. Dr. A. D. Ruedemann, Cleveland.

Frequently it is found that patients who are examined for glasses have a muscle imbalance which may be due to hypothyroidism or other glandular dysfunction. Hyperthyroidism produces definite eye changes, which in most cases are benefited by operation, namely, wide fissures, ulcers and exophthalmos. Associated muscle changes are little benefited by any treatment, medical or surgical. In parathyroid tetany, lens changes are sometimes present, probably the result of a combination of spasm with a deficiency of calcium and phosphorus. Dysfunction of the pituitary gland is a causative factor in certain retinal disturbances and is an associated factor in other eye changes, probably of polyglandular origin.

The recent work of Dr. Crile also brings out a group of cases in which suprarenal dysfunction is associated with eye changes.

(This article will appear in full in The Journal of the American Medical Association.)

DISCUSSION

Dr. William Zentmayer, Philadelphia: Friedgood, Sattler, Barker and Haines have all pointed out that the eye signs hitherto considered classic indications of exophthalmic goiter have been frequently noted in many other diseases. Friedgood believes that the ocular manifestations are of little value in the differential diagnosis of the disease, but of great importance in indicating objectively the actual state of toxicity in the sympathetic nervous system. It would therefore appear that to prove that because the patient has endocrine disturbance the eye symptoms are due to deficiencies of the internal secreting glands, the other conditions enumerated, in which the exophthalmic goiter syndrome may be present, must first be excluded.

Dodds, a biochemist, stated that the view that the exophthalmos is due to the internal secretion of the thyroid and is in some way connected with the amount of thyroid secretion in the blood, is not true. He said that if the basal metabolic rate is estimated throughout a case of exophthalmic goiter, one can tell fairly accurately how ill the patient is, but it is impossible to correlate the metabolic rate determined with the degree of exophthalmos.

In view of the danger of postoperative tetany and cataract, it is interesting to note that irradiation of the thyroid in hyperthyroidism has shown favorable results.

The author's finding that all cases of unilateral exophthalmos were cases of pseudo-exophthalmos due to unequal or unilateral retraction of

the lid will require confirmation in the light of the several cases of unilateral exophthalmos in exophthalmic goiter previously reported.

Contrary to the opinion of the author, it is probable that tetany is due to parathyroid deficiency. It is doubtful whether operations on the thyroid are of much value in materially reducing exophthalmos.

DR. T. B. HOLLOWAY, Philadelphia: One hundred patients with exophthalmic goiter were examined in the hope of discovering some relation between the thyroid and pituitary glands. We found none. In one case seen later such a relationship was definitely suggested.

What method did the essayist use to determine exophthalmos?

In regard to cases of pituitary disease in which there is diplopia without associated muscle palsy, certain English investigators called attention to cases of miners' nystagmus with binocular single vision and yet with better perimacular vision than macular vision, the conclusion being that although the eyes oscillated there was no one point where vision was better than at any other, and thus no diplopia developed.

They then called attention to the fact that in certain pituitary body cases in which the macula is involved and there is central scotoma, in all probability the extra-ocular muscles call into play points on the retina not previously associated, and as a consequence there is diplopia without ocular muscle involvement.

DR. DANIEL B. KIRBY, New York: There is no evidence of any parathyroid deficiency in senile cataract. Calcium metabolism has been normal in carefully observed cases. No benefit was observed in patients with cataract to whom parathyroid extract-Collip was administered.

Lens epithelium grown in vitro will not stand reduction of available calcium without cellular damage. There is undoubtedly a reduction of the available calcium in the lens in parathyroid deficiency, but its exact significance is hard to say.

Apparently, arrest of the cataract process, once started, is difficult even though the calcium balance be restored and the convulsions stopped. Prevention is important and all postthyroidectomy cases should be watched for parathyroid deficiency.

Wheeler's method of shortening and narrowing the palpebral fissure is very satisfactory in those cases of exophthalmos requiring protection of the globe.

DR. A. D. RUEDEMANN: There were in our series 150 cases of postoperative tetany, and 18 patients had cataracts that had been overlooked. They came in shortly after the operation and spoke of a sudden blurring of vision that lasted for a minute the first time and increased in duration until it lasted for half an hour. Observation revealed a slight amount of granular deposit on the posterior capsular surface. This increased and as the spasms increased these flecks assumed the line of contraction of the lens. It is my assumption that they were spicules, and as the spicules became more apparent the lens changes became more diffuse, and these patients developed complete cataract, often in three weeks.

In regard to pituitary involvement, as I said at the outset, one cannot isolate a single gland. If there is hypothyroidism and one looks for changes in the pituitary gland, one will usually find them. These patients usually have increased dextrose tolerance without any changes in the blood. If the general condition does not have anything to do with the

eye condition, surely treatment is not of value. However, if the patients are given thyroid or pituitary extract they get over most of their difficulties.

Patients with hypothyroidism, pituitary dysfunction and high myopia have a low basal metabolism, even as low as —51. They have a flat dextrose tolerance curve, and they are definitely helped by thyroid extract given over a long period of time. This is also true of cases of keratoconus. Most patients with keratoconus have hypothyroidism, but they change from that class to hyperthyroidism.

I am sure that the more one observes these cases the more one will see that there is some connection between endocrine disturbance and eye disease.

PRACTICAL POINTS IN OPHTHALMIC PRACTICE: A STUDY OF RECENT FOOD RESEARCHES. DR. LAURA A. LANE, Minneapolis.

Evidence is set forth showing that one of the most constant signs of food deficiency is pigmentation of the conjunctiva and reduction of the light sense. The retina stores vitamin A, and an avitaminosis causes an increase of lipoids in the rods of the retina and decreases the visual purple. Lack of vitamin A has been responsible for epidemics of hemeralopia, xerophthalmia and keratomalacia. Vitamin A is largely stored in the liver and is much depleted in chronic illnesses; it appears to be concerned largely with preventing infection. Vitamin B is necessary to good nutrition. Lack of it causes nervousness and irritability, and the patients complain of the eyes tiring easily. The addition of vitamin B concentrate to the diet of those suffering from an obscure uveitis of unknown etiology is beneficial. Increasing evidence shows that vitamin C is often lacking in the diet. A lack of vitamin C, combined with a calcium deficiency, may be responsible for repeated vitreous hemorrhages of unknown etiology.

The mineral salts are potent substances concerned in regulating the physiologic processes of the body. Calcium is more frequently deficient in American diets than any of the other mineral elements. Calcium deficiency occurs in vernal conjunctivitis. The complexity of the protein constituents of foods is shown. Proteins of poor quality can cause ophthalmia. The consumption of carbohydrates is often two or three times the amount required for good nutrition. The excessive use of carbohydrates has been known to cause inflammatory diseases of the eye. Excessive fat diets deficient in mineral elements and vitamins cause hyperplasia of the tissues. The problem of food intake concerns itself with sufficient mineral elements and protective substances to maintain proper equilibrium of the body fluids and tissues.

Tables showing the various mineral constituents of foods, the protein, carbohydrate and fat content of the edible portions and the vitamins are given in the author's reprints (see also *Transactions of the Section on Ophthalmology of the American Medical Association,* 1931).

(This article will appear in full in THE JOURNAL of the American Medical Association.)

DISCUSSION

DR. ALBERT N. LEMOINE, Kansas City, Mo.: Until we know more about the biochemical processes that take place in the various obscure eye

diseases, we should avail ourselves of what is known about biochemical processes and their controlling forces—the endocrine system—and regulate them as far as possible with the necessary endocrine therapy and a diet high in its content of all of the vitamins with a proper balance of minerals, carbohydrates, proteins and fats.

DR. I. S. TASSMAN, Philadelphia: From a practical standpoint, it is no doubt true that in many patients suffering from ocular conditions, there exists a deficiency, or at least a partial deficiency, of some of the essential food factors, particularly vitamin A. In the management of such conditions as chronic blepharitis with crust formations on the lid margins, the ordinary subacute and chronic forms of conjunctivitis, recurrent hordeolum and chalazion, phlyctenular conjunctivitis and keratoconjunctivitis in children, episcleritis and simple ulcers of the cornea, a detailed inquiry into the diet of the patient to obtain exact knowledge of the foods included and those omitted will often reveal a genuine deficiency, which if corrected will do more to correct the ocular disturbances than the local treatment ordinarily used.

SOME ANOMALIES OF THE FUNDUS: STEREOSCOPIC PHOTOGRAPHIC DEMONSTRATION. DR. ARTHUR J. BEDELL, Albany, N. Y.

Attention is called to the great number of developmental changes found in the fundus. After a consideration of the variations in the size and surface of the disk, colloid bodies are illustrated with sufficient clinical description to assist any ophthalmoscopist in making a correct diagnosis. The epipapillary membranes are described as of three classes: ragged masses, glistening membranes and persistent hyaloid tubes. By structure and location all of these are proved to be incomplete absorption of embryologic tissue.

Medullated nerve fibers are evident either as massive, glistening white patches, usually about the disk border, or as fine linear striations, sometimes remote from the nerve head. When the medullation is thin, the covered nerve fibers can be traced in their arcuate course.

The pigmentation of the fundus is variable. There may be a complete absence of pigment, as in total albinism, or localized pigment defects, as in partial albinism. These and congenital collections of very dark chocolate spots are described.

Reference is made to the vascularization of the fundus, and some types of vessel distribution are illustrated. The cilioretinal artery and vein, opticociliary vein and choroidovaginal vein receive special mention, the first because of its great importance in embolism and the last because it is often mistaken for a hemorrhage.

Finally, the colobomas are photographed to show the structures involved in the defects and also to demonstrate the distinction between inflammatory reactions and congenital malformations.

(This article will appear in full in THE JOURNAL of the American Medical Association.)

DISCUSSION

DR. ROBERT VON DER HEYDT, Chicago: Moderate variations from the normal in the contour of the nervehead are often atypical colobomas. Instances are common in which inflammatory lesions, often peripheral,

have been accompanied by exudative outpourings into the prepapillary spaces. These exudates may later simulate membranes of glial tissue origin.

An eye presenting a congenital fundus anomaly may also be involved by inflammatory processes. I would not so readily attempt to differentiate between hyaloid remnants near the papilla and extensive processes of so-called retinitis proliferans in the same eye.

The author's statement that Cloquet's canal also shrinks until it ruptures, most often a short distance back of the posterior surface of the lens capsule where a small remnant is almost invariably found with the slit lamp, is confusing the hyaloid artery remnant with Cloquet's canal. The latter anteroposterior embryonic space through the vitreous cannot shrink and in any way become visible.

CHOROIDEREMIA: REPORT OF A CASE. DR. WALTER R. PARKER, Detroit, and DR. F. BRUCE FRALICK, Ann Arbor, Mich.

This article with the discussion appeared in full in the ARCHIVES OF OPHTHALMOLOGY for August, 1931, p. 213.

THE MECHANISM OF ACCOMMODATION: FACTS AND FANCIES. DR. W. H. LUEDDE, St. Louis.

This article with the discussion will appear in full in a later issue of the ARCHIVES OF OPHTHALMOLOGY.

NORMS OF REFRACTION. DR. EDWARD JACKSON, Denver.

This article does not lend itself to abstraction, and should be read in the original. It will appear in full in THE JOURNAL of the American Medical Association.

DISCUSSION

DR. DAVID W. WELLS, Boston: I shall confine my discussion to the development of senile cataract. Opinions differ as to the value of treatment in this condition, and the truth will not be known until the natural history of this disease is compiled. In 1915 I found that, in a series of cases followed from ten to fifteen years, if the vision at the time was 0.5 or better, not more than 5 per cent had actually advanced.

If we all possessed the cataract norm based on thousands of cases tabulated by fifty or a hundred men in different parts of the country, we might tell the patient the truth and prevent his fears by showing him the record. One recent writer has justified his continued treatment by saying: "It is salutary and reassuring to the average patient to offer hope of arresting a condition which he regards as hopeless." This is probably the attitude of many who persistently treat these patients, and if the case does not progress, allow them to believe that the treatment has retarded it. In the words of Vogt: "The road to Charlatanry is not far off."

DR. J. MONROE THORINGTON, Philadelphia: Percentage tables are only relative, due principally to the concentration of abnormalities in a physician's practice. A more general study of emmetropia will doubtless increase considerably the percentage falling into this category.

The astigmatic group affords an especial opportunity for study. Dr. Jackson divides it into the horizontal, vertical and oblique types. Our belief is that additional study will further subdivide these, bringing

out evidence of axial symmetry in the two eyes of many patients, and pointing to the extreme rarity of certain oblique meridians.

Dr. Peter C. Kronfeld, Chicago: The problem of normal refraction is a puzzling one. Dr. Brown and I, in Chicago, tried to determine the relative frequency of every refractive state. In 10,000 patients whom we examined under the influence of atropine we listed the two main meridians as units of refraction, and by doing so we obtained what is usually called the normal refraction curve. The greatest frequency falls on a point that corresponds to a refraction of 31.1 under atropine, and from there the curve slides down gradually on each side, both the myopic and the hyperopic side.

The same curve is compared with the so-called normal frequency curve, which can be applied to most of the phenomena of nature. The normal frequency curve was found first by a mathematician named Gaus. The human refraction curve differs from it in several respects. From a study like this one would conclude that the most frequent refraction under atropine is 1.25; without atropine, +1.75. I should think that the most frequent refraction is the normal refraction.

Dr. Edward Jackson: With reference to some statistics that have been alluded to by Dr. Kronfeld, on seeing these curves when the paper by Dr. Brown and Dr. Kronfeld was first published I was struck with the close resemblance to what I had observed in a much smaller number of eyes, 4,000 instead of 10,000, when we found the greatest number had between 1 diopter and 1¼ diopter of hyperopia. I think that we can understand why the curves have a frequency of different amounts of ametropia that do not correspond to the curve of Gaus. It is because in our hustle and hurry today, the easy readjustment of the eye on a focus is becoming so important that those who do not have it and cannot easily adjust for the ordinary use of the eye are left out. These high degrees of hyperopia and myopia are therefore disappearing from our statistics.

The Pocket Flap: The Safest Method for the Extraction of Senile Cataract. Dr. Charles Nelson Spratt, Minneapolis.

A horizontal incision, from 1.5 to 2 cm. long and 5 mm. above the limbus, is made in the conjunctiva, and dissected down to the limbus, the upper two fifths of the corneal circumference being freed. A mattress suture is placed in the center of the edges of the conjunctival wound. Fixation is by the conjunctival flap; the incision is made at the limbus. The advantage of this subconjunctival incision is that it provides (*a*) the least chance for infection, (*b*) less danger of vitreous escape and (*c*) a suture. A sclerectomy may be done at the same time as the extraction. Healing takes place in a few hours. There is no iris prolapse. The patient may sit up immediately after the operation.

DISCUSSION

Dr. William A. Fisher, Chicago: Suggestions to be added to every operation for senile cataract to make the pocket-flap operation safer would be: 1. Draw off 8 ounces (226.8 cc.) of blood from a vein, as Professor Elschnig does, one hour before operating, provided the blood pressure is over 180. 2. Use injections of procaine hydrochloride for relaxing the orbicularis muscle, and use deep orbital injection of the same drug to reduce ocular tension. 3. Place a suture in the superior

rectus muscle to aid in making the toilet when necessary. 4. Use lid hooks instead of the speculum. 5. Make a full or peripheral iridectomy. 6. Make an effort to remove the lens in its capsule. 7. Close the lids for twenty-four hours with a skin suture loosely tied. 8. Open the operated eye less often for a dressing. 9. Do not see how quickly a patient can leave the hospital.

DR. SAMUEL G. HIGGINS, Milwaukee: The sutured pocket flap is more exact in its application than the old conjunctival bridge or loose flap. Hemorrhage occasioned by a wide cut through the conjunctiva or by undermining a width of several millimeters of conjunctiva, especially when the episcleral tissue is included, may sometimes be annoying if not serious. Increased rather than impaired visibility of the field of operation is desired. Two disadvantages mentioned by the author are the difficulty of making the incision and the deep separation of the edges of the wound. The dissection of the flap down to the upper two fifths of the cornea may not permit as wide an incision as some surgeons are in the habit of making. Admittedly, conjunctival covering of the wound does not prevent deep separation of the wound.

HYPERGLYCEMIA IN PERSONS WITH ADVANCED SENILE CATARACT. DR. C. S. O'BRIEN, Iowa City.

In this study an attempt was made to determine the relationship, if any exists, between hyperglycemia and senile cataract. Knowing that changes in the lens occur as a complication of diabetes and other metabolic diseases, it was thought that hyperglycemia, if it existed in those affected with senile cataract, might have some part in causing these changes. If there existed even a moderate increase in the sugar concentration of the blood and of the fluids bathing the lens, and this acted over a period of years, possibly there was a deleterious action on the cortical fibers of the lens. It really would not matter whether the patient had clinical diabetes or not; so long as a hyperglycemia existed, such an action could take place.

The study was made on a series of 238 patients with senile cataract as they were admitted to the wards of the hospital for extraction of the lens. The age of these subjects was from 38 to 91 years, 95 per cent of them being between the ages of 50 and 88. The average age was 66 years. Determinations of the fasting capillary blood sugar were made by the Boyd micromodification of the Folin-Wu method on 238 patients. In 99 cases (41.6 per cent) the fasting blood sugar was above the recognized maximum normal limit of 120 mg. per hundred cubic centimeters.

It is interesting to note that in this series there were only twelve patients with advanced diabetes, i. e., patients who had already had a clinical diagnosis of diabetes.

Hyperglycemia is occasionally associated with arteriosclerosis, advanced kidney lesions and other diseases, and this may have accounted in part for the high percentage of cases showing abnormally high sugar concentration. But from a study of the records of the systolic blood pressure it may be seen that there were few with hypertension. Also the cases presenting advanced nephritis were not numerous.

The dextrose tolerance test was also utilized as a method of determining the ability of these patients with cataract to utilize carbohydrates. Curves have been established for normal fasting subjects. If the maximum concentration of blood sugar is above 200 to 220 mg., or the peak is not reached within thirty to sixty minutes following the ingestion of a 50 Gm. dextrose meal, or the blood sugar does not return to normal within two and one-half hours, the curve is considered abnormal by most investigators. Prolonged over a period of years, this condition may have an effect on the lens. In this series of 214 senile cataract cases the maximum hyperglycemia was above 200 mg. in 129 curves, above 220 mg. in 99 and above 240 mg. in 65. The peak was reached in sixty minutes or more in 166 of the 214 cases; it was reached in ninety minutes or more in 40 cases. The blood sugar had not returned to normal within two and one-half hours in 96 of the 214 cases, and in 41 cases it had not returned to normal within three or more hours. Thus it may be seen that the normal sugar level was disturbed in approximately half of the cases. In those of my patients with a low tolerance, there occurred repeatedly a hyperglycemia, and the lens was subjected to higher concentrations of sugar in the surrounding fluids.

(This article will appear in full in THE JOURNAL of the American Medical Association.)

DISCUSSION

DR. DANIEL B. KIRBY, New York: It is important to compare the venous with the capillary blood sugar if we wish to know the full story of carbohydrate metabolism. A study by one of my associates, Renee Wiener, has been the behavior of the blood sugar after the injection of insulin into these patients with cataract. The sugar depletion curve produced thereby is nearly always a mirror image of the dextrose tolerance curve.

I have been making a number of tests to determine the relationship between the aqueous sugar and the blood sugar both in patients with known diabetes and incipient lens opacities and in patients with cataract who have had no symptoms of diabetes. Increases or decreases in the blood sugar are not immediately reflected in the aqueous sugar. Undoubtedly due to the slowness of diffusion into the lens itself, the time lag is still greater, so that the responses manifested by changes in refraction come some time after the concentration of sugar has changed in the blood.

In the tissue culture experiments we have found that crystalline lens epithelium grown outside the body will utilize relatively very high sugar concentrations. I surmise that the deleterious action of increased sugar in the aqueous and lens is in the sensitive colloid of the lens protein. The vacuolation and degeneration of the pigment epithelial layer in diabetes extends well back into the ciliary epithelium and as such will surely affect the quantity and quality of the aqueous formed. This factor may be responsible for the development of the lens opacities in diabetic patients.

The principal thing to consider is whether the decreased dextrose tolerance is the cause or the result of the main disease, or whether it is simply a coincidence. If it is the cause, the proper procedure is to treat the patient for latent or potential diabetes.

DR. ALAN C. WOODS, Baltimore: Dr. O'Brien brings to the front the interesting suggestion first brought forward over a half century ago by Weir Mitchell, and strengthened by the well known experiments of Schanz, that perhaps in the human being small changes in the sugar content of the body fluids may have either a direct action in the production of lens opacities, or else may act as a contributory factor—e. g., causing changes in the electrolytic content—and thus allow other agents to produce lens opacities.

Dr. O'Brien's evidence should be very carefully examined. After a dextrose meal, 240 rather than 220 mg. should be regarded as a high normal figure. Of the 238 cases presented by Dr. O'Brien, 99, or almost 42 per cent, showed a fasting blood sugar of over 120 mg. per hundred cubic centimeters. An analysis of Dr. O'Brien's figures concerns itself solely with these 99 patients. Dr. O'Brien states that there were 12 clinically diabetic patients in this series. By this I should infer that he means that there were only 12 patients who had a glycosuria, changes in the carbon dioxide-combining power or evidence of diabetes other than the fasting blood sugar. On examining his tables, one finds there were 3 patients with a fasting blood sugar of 202+ mg. per hundred cubic centimeters, 21 with a fasting blood sugar of 217— mg. per cent and 35 with a fasting blood sugar of 182+ mg. I cannot but believe that these 59 patients must have true diabetes, although only 12 are so diagnosed clinically. To my mind, these 47 patients have the most dangerous type of diabetes —with a high renal threshold for sugar—the type of diabetes that is most liable to the various complications of the disease, of which the formation of cataracts is only one. Before we draw any conclusions from these figures, I think that these 59 patients should be discarded from the series, for they appear to be patients with ordinary diabetic cataracts, and the results of chemical analysis in such cases cannot be used to support the premise that a low grade hyperglycemia in nondiabetic patients may be a contributory cause to the development of cataracts. While Dr. O'Brien's figures, with all presumably diabetic persons discarded, still show 22 per cent of nondiabetic cataractous patients with a moderate hyperglycemia, when we consider that practically all of these hyperglycemias occur in elderly individuals liable to develop arteriosclerosis and that the estimations were made by the Folin-Wu method (which gives higher readings than the methods that use the actual fermentation of sugar as their basis), I do not believe that these figures vary much from what we would normally expect in a similar number of noncataractous persons. When we consider the dextrose tolerance test, we find very much the same situation.

Summing this up, in this series of 238 patients there were 59 who I believe should be discarded as diabetic. This leaves 179 patients from whom conclusions can be drawn. Among these 179 patients there were 39 elderly persons, or 22 per cent, who had a moderately elevated fasting blood sugar, and probably about 18 patients, or 10 per cent, who showed an abnormal blood sugar content after a dextrose meal. When we consider the age of these patients, the high frequency of arteriosclerosis in elderly persons and its influence on the blood sugar picture, these figures to me are far from convincing.

The proposition advanced by Dr. O'Brien is, I think, of the greatest interest, even though I cannot agree that his figures present any confirmatory evidence.

DR. H. MAXWELL LANGDON, Philadelphia: In the series of cases that I have followed the blood sugar was found to be high, and in 65 per cent the sugar tolerance test was distinctly abnormal. The sugar tolerance test is very useful. Carbohydrates form a much larger part of our diet now than in older times. In experiments with dogs, monkeys and fowls the blood sugar was much higher in the animals with cataract than in the normal ones.

DR. C. S. O'BRIEN: I do not agree that 240 mg. should be accepted as the upper limit of the result for the dextrose test.

I question the discarding of the 59 patients which Dr. Woods mentions. That can be done only when we are considering whether or not these patients show a hyperglycemia. It is not a question of diabetes; it is a question whether increased circulatory sugars have any effect on the translucency of the lens, and certainly these 59, who had not been classed as diabetic and had not been on diabetic management, must be included because of the fact that hyperglycemia existed.

POSTOPERATIVE NONEXPULSIVE SUBCHOROIDAL HEMORRHAGE. DR. BERNARD SAMUELS, New York.

This article with the discussion will appear in full in a later issue of the ARCHIVES OF OPHTHALMOLOGY.

INTRA-OCULAR HEMORRHAGE FOLLOWING OPERATIONS ON THE GLOBE. DR. HARRY W. WOODRUFF, Joliet, Ill.

Five cases from the laboratory of the Illinois Eye and Ear Infirmary, comprising four cataracts and one glaucoma, showed extensive disease of the choroid and retina. Also there was marked sclerosis of both arteries and veins with areas of round cell infiltration.

The important etiologic factors in the production of cataract and glaucoma may also be predisposing factors in the occurrence of the type of hemorrhage under consideration. Degenerative vascular changes accompany advancing age; also the presence of toxic conditions, syphilis, nephritis and alcoholism, with their predilection for the vessels, causes either primary or secondary endarteritis and vascular sclerosis.

It is not at all surprising, then, that some of the less violent forms of intra-ocular hemorrhage occur in 4 or 5 per cent of operations for cataract and glaucoma; when the larger vessels, such as the long posterior ciliary arteries, are affected, an expulsive hemorrhage occurs in from 0.01 to 2 per cent.

DISCUSSION

DR. WILLIAM H. WILDER, Chicago: The etiology of most of these unfortunate cases, both severe and mild, is probably to be found in a condition of atheroma of the arteries of the retina, uveal tract or long ciliary vessels brought about by syphilis, renal disease, diabetes or the natural involution of old age. Some may be due to hemophilia or some blood dyscrasia not easily determined.

In regard to the cases reported by the essayist, I think that his comments are wise. I think that it is not best to operate on both eyes at the same session when the eyeball is to be opened or penetrated, as in his

first case. One does not always know when a serious complication is present or when an accident is impending, and it is better to be safe than sorry.

In this connection I want to refer to, and emphasize the importance of, careful protection of the eye after the operation. Probably if the aggregate experience of all operators could be gathered and tabulated it would be found that an unnecessarily large number of intra-ocular hemorrhages and injuries of the eye occur from the neglect to use a well fitted protective shield, such as the Ring mask, or the simple expedient of properly securing the patient's hands so that he cannot get them up to his eyes when he is asleep or even dozing.

Directory of Ophthalmologic Societies *

FOREIGN

British Medical Association, Section on Ophthalmology

President: Mr. E. E. Maddox, Glenartney, Poole Road, Bournemouth, England.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 7-9, 1932.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Paris. Time: July 17-21, 1932.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Leipzig. Time: June, 1932.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Rotterdam, Holland.
Place: Madrid, Spain. Time: 1933.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: New Orleans. Time: May 9-13, 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. F. Barnhill, Miami Beach, Fla.
President-Elect: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Montreal, Canada. Time: 1932.

American Ophthalmological Society

President: Dr. Edward C. Ellett, 130 Madison Ave., Memphis, Tenn.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.
Place: New London, Conn. Time: June, 1932.

SECTIONAL

New England Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Me.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. Claude T. Uren, City National Bank Bldg., Omaha.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Fontanelle Hotel, Omaha. Time: November, 1931.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. C. Ward Ellis, 215 N. Walnut St., Lansing.
Secretary: Dr. H. B. Weinburgh, 301 Seymour St., Lansing.
Time: Third Thursday of alternate months.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the Month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. W. O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Gordon F. Harkness, 215 Main St., Davenport.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Douglas F. Wood, 74 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: W. L. Diven, National Bank Bldg., Bismarck.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. M. Hendershott, 193, 11th St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. R. Thompson, 600 W. 10th St., Fort Worth.
Secretary: Dr. A. F. Clark, 414 Navarro St., San Antonio.
Place: San Antonio. Time: December, 1931.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. Franklin H. Raley, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. H. L. Smith, 1005 Medical Arts Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. C. P. Jones, Newport News.
Secretary-Treasurer. Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Charles W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Earl LeRoy Wood, 192 Roseville Ave., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John R. Childs, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Alan C. Woods, 906 Cathedral St., Baltimore.
Secretary: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. A. F. Luhr, 40 W. North Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Roy B. Metz, Hanna Bldg., Cleveland.
Secretary: Dr. Paul G. Moore, Medical Arts Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadephia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Ivor Clark, 188 E. State St., Columbus, Ohio.
Secretary-Treasurer: Francis W. Thomas, 327 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. M. Sellers, 717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831, 5th Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. J. J. Richardson, 606 Penn St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, 1304 Medical Arts Bldg., Forth Worth.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robison, Eye, Ear, Nose and Throat Hospital, Houston.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. R. R. Montgomery, 102 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Russell T. Uhls, 823 Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Oto-Laryngology

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Herbert F. Wolters, 130 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. William P. Haney, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. A. Cassiday, 1620 Medical Arts Bldg., Omaha.
Place: Medical Tea Room. Time: 5:15 and 7:00 p. m., third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Emanuel U. Wallerstein, Professional Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Martin H. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott C. Applewhite, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. W. F. Swett, 490 Post St., San Francisco.
Secretary: Dr. Robert C. Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. Claude L. La Rue, Highland Clinic, Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse.
Place: University Club. Time: Second Friday of each month except June, July and August.